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An Address.[1]

SOME ASPECTS OF PUBLIC HYGIENE AND HEALTH ADMINISTRATION IN SOUTH AUSTRALIA.

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As the President of this Branch of the British Medical Association vacates his position it is customary for him to give an address.

What the subject of that address will be is entirely left to himself, and he naturally chooses one in which he is practically interested or a knowledge of which may be of some benefit to his fellow practitioners.

[1] Delivered at the annual meeting of the South Australian Branch of the British Medical Association on June 25, 1931. Received for publication September 7, 1931.

I taxed my memory as to what others have done under similar circumstances, and, looking back over the last five years, find that the addresses have been decidedly varied, and for that reason have chosen one of that type also and which concerns more or less the whole profession.

In dealing with the subject of "Some Aspects of Public Hygiene and Health Administration in South Australia," might I be permitted to go back to the period when Australia was first settled, and I cannot do better than quote a paragraph from the present chief of health administration in Australia.

In THE MEDICAL JOURNAL OF AUSTRALIA of April 25 last Dr. J. H. L. Cumpston, Director-General of Health, Commonwealth Department of Health, states in summing up his review of the first forty-two years of public health in Australia:

This introductory period of Australian history presents to the student of public health an absorbing picture show-

ing progressive presentation in each of the major public health problems. A community taken for the most part from a grossly insanitary environment in England, infected with typhus fever, cholera, dysentery, typhoid fever, tuberculosis and venereal diseases was landed after an eight months' voyage in a country free from all diseases or unhygienic conditions. The community was under the control of a governor whose commission contained no reference to the health of the people entrusted to his care. This community experienced scurvy from lack of proper food, carelessly polluted its own water supply, ignored exhortations to cease such pollution and compelled the enactment of increasingly stringent legislation designed to prevent this danger to their health which, of their volition and by their own self-restraint, they should have made impossible . . . . On various occasions vessels arrived carrying infection from other countries. An ordinance prescribing certain quarantine measures was slavishly copied from English precedent, without any local application. Later imminent danger from infected vessels and the concealment by the ship's officers of the occurrence of infectious disease on their vessels, resulted in the application of rational quarantine measures.

Since the establishment of departments of public health in the various States and the advent of the Commonwealth in the field of public health, the legislation that has been enacted, together with the care and tact of its enforcement, has resulted in the prevention of the suffering of individuals, the saving of lives, the purity of food supplies and vastly improved housing and other conditions which the community in all walks of life now enjoy.

The money that has been expended is almost negligible when one considers the benefits that have accrued and the improvements effected in the various directions.

> Gold that buys Health can never be ill spent,
> Nor Hours laid out in harmless merriment.
> (John Webster—"Westward Ho!")

I am indebted to the "Municipal Year Book" of the City of Adelaide for the following information with regard to the history and public health administration in South Australia.

The evolution of the health legislation of South Australia is interesting in so far as each health act reflects the scientific knowledge of its time.

The first comprehensive Health Act was passed in 1873. It was based on recent English legislation following a sanitary awakening in that country and the result of repeated outbreaks of cholera.

It established a central board of health and constituted the council of each town a local board of health. This Act dealt almost exclusively with the abatement of nuisances. One or two clauses referred to "Epidemic, Endemic and Contagious Diseases" generally.

The second Health Act, an amending one, was passed in 1876. It advanced a stage by recognizing that the destruction of bedding, clothing and other articles which had been exposed to infection from the "Epidemic, Endemic and Contagious Diseases" might prevent the spread of such diseases.

The third Health Act, passed in 1884, was also an amending one. The amendments dealt almost exclusively with infectious diseases and provided for notification, isolation and disinfection, but the diseases were limited to smallpox, cholera, plague and yellow fever.

As these were all exotic diseases, the provisions of this Act were useful only when a case had escaped the vigilance of the Quarantine Department. Still, the Act recognized the fact that infectious diseases could not be checked by exclusive attention to insanitary conditions, but called for special consideration. The time for applying this consideration to infectious diseases generally had not yet arrived.

The present position of public health in this State has been gradually evolved from experience in the British Isles, in the older States of Australia and other parts of the world.

The greatest work done by legislation was in the year 1898, when the last or fourth *Health Act* was passed (amending Acts were added in 1909, 1911, 1916, 1918 and 1925) and it was contemporaneously with this 1898 Act that the medical profession had their largest representation in the State legislature, and it is in this connexion that I might mention the names of the late Sir John Cockburn, Dr. S. Magarey and Dr. Alan Campbell.

When the *Health Act*, 1898, was being framed, certain sections in Part 8 relating to the control of infectious diseases and pulmonary tuberculosis were known as the "Campbell Clauses." At that time in no other health act perhaps did similar clauses exist, but Dr. Campbell, watching from afar the inception of the great movement directed against tuberculosis and to a certain extent infectious diseases, and realizing what was wanted in the Old Country legislation and what would materially assist this State, exerted his great personal influence and authority to secure the inclusion of these clauses in the Act.

The idea at that period was to bring about all the available legal and administrative experience gained in the old world and so to study public health problems under new conditions while keeping in mind all the well recognized practices that are necessary for public safety.

Of the population of South Australia 55·73% is resident in the metropolitan area and the rest of the State is sparsely populated. (The State population is 582,127 and the metropolitan 324,420.)

The health administration of the State is founded on the principle of local government with a central authority, "The Central Board of Health." The Acts administered are: *The Health Act*, the *Food and Drugs Act*, and the Act dealing with the early notification of births.

The Minister controlling health matters is the Honourable the Chief Secretary.

The Central Board consists of five members, three of whom (including the Chairman, who is also the permanent head of the department) are appointed by the Governor and the remaining two are elected bi-annually, one by the city and suburban local boards of health and the other by all other local boards. Each municipal and district council is the local board of health for its district, the members being elected by the ratepayers. There are 197 local boards in the State.

Local boards are charged with the due execution of the *Health Act* for securing the proper sanitary condition of their respective districts and under the *Food and Drugs Act* for maintaining pure food supply.

The Central Board acts as a supervisory body under both the *Health Act* and the *Food and Drugs Act*, and supplements this by an active policy of

inspection and advice and also has concurrent jurisdiction with local boards. Where there is no local board, the Central Board alone administers under both the *Health Act* and the *Food and Drugs Act*. The Central Board has power to supersede the local board in the event of default of action by such board and to recover all reasonable expenses that may be incurred.

The Central Board has two male inspectors, who hold the certificate of a recognized sanitary institute. They periodically visit the local board districts with the view of ascertaining whether they are carrying out their duties. It also has a nurse inspector, who holds the certificate of the Royal Sanitary Institute and is certificated in nursing and midwifery. She advises and assists local boards in connexion with the prevention and control of outbreaks of infectious diseases and the conduct of private hospitals and maternity homes and also carries out certain duties similar to those performed by the male inspectors.

Municipal and district councils have power to declare such rates as may be necessary for expenditure by the local board for the purposes of carrying out the provisions of the *Health Act* and may also under certain circumstances expend from the general revenue money for such purpose without declaring a rate.

It is incumbent upon local boards to appoint an officer of health who, where practicable, must be a legally qualified medical practitioner. His appointment and dismissal are subject to the approval of the Central Board of Health. In addition to the duties assigned to him, he possesses all the powers and duties of an inspector under the Act. All the officers of health are legally qualified medical practitioners. Some of the local boards, having extensive districts, have more than one such officer. As regards inspectors, the Central Board may require local boards to appoint such as may be necessary or desirable. Usually the secretary or overseer of works fills the office in country districts. The inspector has very extensive powers. He may enter and inspect premises, Government and otherwise, at convenient times, and in the event of his being obstructed in his duties, may invoke the assistance of the police. Consequently the effective administration of the *Health Act* depends a great deal on the training, character and general work of the inspector. He is always required to carry out any directions he may receive from the officer of health.

Certain local boards possess the services of a trained nurse inspector who is certificated in sanitary inspector's work and also in nursing and midwifery.

Local boards are required to keep separate accounts of their revenue and expenditure and minutes of their proceedings.

If an inspector of a local board discovers the existence of an insanitary condition, he should furnish a report on it to his board. If the inspector be of opinion that the insanitary condition should be immediately removed, he should forthwith serve a notice for its removal or amendment and report the circumstances to his board, which has power either to adopt the notice to give it legal effect or to issue a new notice in lieu thereof.

As regards insanitary conditions beyond those requiring immediate attention, which may be reported to the local board, such board is in power to declare such to be an insanitary condition and to serve notice for its removal or amendment, specifying a time for compliance. If the notice is not complied with within the time specified, the local board may take legal proceedings for such failure.

A branch of the Royal Sanitary Institute, London, has been established in this State. It conducts examinations for efficiency as sanitary inspectors, for meat inspectors, and in sanitary science. Classes of instruction are held at the School of Mines, and practical experience is gained by candidates accompanying inspectors on inspections and other work.

The Regulating Influence of the Health Act.

Most, if not all, communicable diseases affecting the State can be successfully controlled or prevented, provided that the local authorities make use of the machinery provided by the *Health Act*.

The *Health Act*, 1898, gave powers to every local board of health to provide for the control of infectious diseases within its bounds in the most comprehensive manner and to defray all the expenses of such control from the sanitary rate it was allowed to levy.

In order to check disease it is necessary: (i) to detect every case of illness in the earliest stage, (ii) to keep separately those that are sick, (iii) to keep under observation contacts of the sick and to isolate them if necessary. The isolation should be sufficient to prevent infected or infective material from passing from the sick to the healthy. The attitude taken up by some individuals and communities to public administration varies from active and loyal cooperation to apathy and even hostility, including misrepresentations of administrative principles. Unfortunately public and private interests may clash, leading to untold troubles in health organization. It should not be forgotten that the greatest national asset is health and not wealth.

In his annual report for 1929 Sir George Newman, Chief Medical Officer of the Ministry of Health of Great Britain, stated that:

> As only an educated people can be an effective people, so also only an enlightened and willing people can be a healthy people.

Infectious Diseases.

Cases of infectious disease must be reported to the local board under the provisions of Part 8 of the *Health Act*, 1898. For this report a fee of two shillings is payable to the medical practitioner by the local board to which he reports the case.

During this year an effort was made by the local boards to do away with the reporting fee altogether. The Government, on the recommendation of the Central Board of Health, disallowed the deletion and the amendment was not carried. Had the amendment been carried, it would have been unfair to medical practitioners, as they would be called upon to expend time and money (postage) with no remuneration and less protection to the general public.

At this juncture it is interesting to state the fees for notification of infectious diseases in the other States: In New South Wales the fee is three shillings and sixpence, in Victoria it is one shilling and sixpence if the case occurs in private practice, and one shilling if it occurs in a hospital or an institution. In Queensland the fee is two shillings and sixpence if the case occurs in private practice, one shilling if in a public hospital or an instituton. No fee is paid in the City of Brisbane. In Western Australia and Tasmania the fee is two shillings.

The duty of giving this notification is generally imposed on the head of the house to which the patient belongs. Failing him, it is imposed on the nearest relative present, and on his default on the person in charge of or in attendance on the patient, and on his default on the occupier of the building; and in addition the medical practitioner must report the case.

No medical practitioner has any right apart from the *Health Act* to give the authorities or anyone else any information about a patient he is attending, and the *Health Act* or the direction of a judge in a court is his only legal protection when giving such information.

Local authorities are required to report from time to time to the Central Board as to the health, cleanliness and general sanitary state of their several districts, and must report the appearance of certain diseases. Power is given for the disinfection and cleansing of premises and for the disinfection and destruction of bedding, clothing and other articles which have been exposed to infection.

Bacteriological examinations for the detection of diphtheria, typhoid and other infectious diseases within the meaning of the *Health Act* and also tuberculosis are continually being carried out.

Visitation and Inspection. As soon as the medical practitioner has reported the case to the local board of health his duty to the public is finished with. His concern is the health of the patient, but it is a good practice for the general practitioner to help further if necessary.

The concern of the officer of health is the health of the community and the prevention of spreading from that particular case to other persons. It is not necessary as a rule for the medical officer of health to see the patient, but should this be so, he must get in touch with the ordinary medical attendant and see the patient with him. At this stage in the procedure promptness is the keynote and isolation and disinfection should be carried out at once.

Home Isolation. Home isolation may be carried out in many instances, particularly in country districts and even in towns and cities, and local boards should always maintain a stock of disinfectants for use either by their officer of health, trained nurse or inspector, and remove for disinfection articles that cannot be properly disinfected at the residence.

When proper isolation is impracticable or when the sick person is lodged in a room occupied by others of more than one family, in a boarding house, the patient can be removed upon the certificate of a legally qualified practitioner and with the consent of the hospital authorities to any hospital for the reception of such patients for the sake of the public health.

Isolation Hospitals. Isolation hospitals are places provided and supported by the ratepayers of a district for their own defence and treatment, and should be open for the reception of everyone in the district who suffers from an infectious disease and who goes there as a concession to the wishes of his neighbours. An isolation hospital or some place to receive and seclude the sick should be in readiness before the necessity to use it arises. If such a place is not provided until the need for it has arisen, it is usually useless as a means of checking the spread of infection and is of use merely for the treatment of particular patients and not for the protection of the community as a whole.

The diseases that are "infectious diseases" within the meaning of the *Health Act* and are reportable, are: Leprosy, plague, yellow fever, smallpox, cholera, diphtheria, membranous croup, erysipelas, scarlet fever, scarlatina and the fevers known by any of the following names or descriptions: typhus, typhoid, enteric, relapsing or puerperal (including all puerperal conditions depending on infection), anthrax, trichinosis, cerebro-spinal meningitis, cerebro-spinal fever, epidemic cerebro-spinal meningitis, intermittent, remittent paludal or malarial fever, whooping cough, measles, tuberculosis in animals, favus, bilharziosis, *influenza vera*, influenza or any febrile toxic septicæmic condition similar to influenza, including pneumonic influenza, chicken pox, *poliomyelitis anterior acuta, encephalitis lethargica* (epidemic encephalitis), ankylostomiasis, amœbic dysentery, bacillary dysentery (filariasis) and paratyphoid fever.

Pulmonary tuberculosis is not an "infectious disease" within the meaning of the *Health Act*, but it is reportable by medical practitioners to the local board of the district in which the patient resides.

Appended is a table of the notifications and deaths of the principal infectious diseases in this State since 1921 (Table I).

Epidemics of Infectious Disease in Schools.

The Central Board of Health has issued publications and pamphlets showing the seriousness and far-reaching effects of communicable diseases among school-going children. In the regulations made under the *Education Act*, 1915, and gazetted on

Table I.

Notifications of and Deaths from Certain Infectious Diseases, 1921-1930.

Year	Diphtheria		Erysipelas		Poliomyelitis		Puerperal Fever		Scarlet Fever		Typhoid Fever		Whooping Cough		Measles	
	Notifications	Deaths	Notifications	Deaths	Notifications	Deaths	Notifications	Deaths	Notifications	Deaths	Notifications	Deaths	Notifications	Deaths	Notifications	Deaths
1921	2744	123	125	8	—	—	22[1]	23[1]	1245	4	245	35	391	22	4560	14
1922	1502	58	106	6	47	—	16	13	1421	3	136	18	54	2	491	4
1923	827	46	102	8	17	—	28	20	1290	9	118	13	929	12	1031	1
1924	657	32	93	9	—	—	27	11	787	4	92	13	1792	52	167	—
1925	407	19	169	7	13	1	62	21	273	2	96	16	174	7	14804	44
1926	557	33	179	12	2	—	63	17	352	3	103	8	58	3	191	—
1927	452	14	172	15	23	3	102	19	418	1	80	16	2905	42	2899	7
1928	374	14	126	5	15	1	86	20	386	4	83	14	574	19	1488	2
1929	329	12	152	4	8	2	73	19	238	—	57	4	497	1	4233	8
1930	245	2	226	12	15	—	75	13	104	—	79	6	4466	38	2466	9

[1] One of these was reported the previous year.

November 18, 1920, the procedure is set forth regarding the exclusion from schools of children who are suffering from an infectious disease or whose presence might be injurious to the health or welfare of other children attending school. If epidemics in schools in this State are to be checked, then concerted and cooperative action is necessary on the part of the Department of Education, local boards of health and the Central Board.

All efforts will be futile if there is any laxity or delay on the part of any one of the responsible departments. Notification has to be prompt, and to secure this a scheme to check such epidemics has been agreed to so far as whooping cough, measles, diphtheria and scarlet fever are concerned. The three parties are to be notified of the occurrence of cases and especially of a "first case" as soon as possible.

A "first case" means one occurring as a new infection, that is, measles three weeks, whooping cough six weeks, and diphtheria two weeks after the death or recovery of the person last reported as suffering from the disease.

The teacher of the school notifies the Director of Education if a school child is taken ill or if there is a case of infectious disease at the house where the school child resides. He similarly also notifies the secretary of the local board of health.

The officer of health should, when a "first case" occurs, visit the premises, collect all particulars required for the special notification form and report to the local board of health the action taken and the instructions given. A copy of the report is sent to the Central Board of Health. The local board of health, on receiving notification from a medical practitioner or other person of the occurrence of infectious disease, should at once notify the Central Board of Health. The Central Board of Health notifies daily the Director of Education the names of all children of school age reported as suffering from an infectious disease, and the Director of Education similarly notifies the Central Board the names of all affected children reported to him.

If it is a "first case," the information should be sent by telegram or telephone and the written notification on the official form (number 3) by post.

All the children who have been contacts are to be excluded from school for such time as is fixed as the limit in the case of the particular disease, for example, measles 16 days, whooping cough 21 days, scarlet fever 14 days and diphtheria 12 days, and they must not return to school unless and until their clothes have been properly disinfected and they produce a medical certificate that there is no risk of infection. Within fifteen days after the date at which a district is "clean" the officer of health transmits through the local board to the Central Board of Health a report of the number of cases occurring in the epidemic, the number of cases terminating fatally, the origin of the epidemic, the means by which the disease was spread and the measures taken to control the epidemic.

Medical Inspection of School Children.

Medical inspection of school children is carried out extensively in this State. Medical staffs have been organized, travelling clinics established to deal with dental, ocular and other defects.

Medical inspection includes the examination of all children attending primary, central, high and technical high schools. Each child is examined once in three years. Any defects likely to interfere with the child's educational progress are furnished by report to the parents. The staff consists of one medical officer, four medical inspectors, one psychologist, one dentist, three trained nurses, two dental assistants and a disinfecting officer. Since 1928 school children are treated at the dental hospitals and dentists attend country schools.

The psychologist examines mentally retarded children and supervises their work in certain classes. During the year 1928 27,443 children were examined by the medical inspectors; of these 631 had defective vision, 202 defective hearing and 1,125 adenoids and enlarged tonsils; 2,328 children received dental treatment.

The Pre-School Child.

Up to recent years there has been the "gap" in the neglected pre-school years between the infant welfare and the school age. These pre-school years, the period of the "toddler" or "runabout," have been slighted or entrusted to the home.

The chief value of medical inspection of school children has been to demonstrate the extent to which children of school age are suffering from defects and disease which might have been prevented or immunized by attention to the pre-school period. Not only is it necessary to study the welfare of the pre-school child, its weight, diet, teeth, adenoids *et cetera*, its physique and also the mental and motor habits. These should not be only parental care, but also educational control. A comprehensive survey of the problem of the pre-school child from the standpoint of public hygiene and education is very essential.

The world war seemed to give an entirely new emphasis to child welfare and child hygiene work. The frightful destruction of human life served to focus attention on the care of the next generation, and in consequence we seem today to be on the eve of a new and very important extension of educational activity, that is, the attention to the child under school age. The enactment of legislation making better provision for school health service in the care of children during pre-school years to eliminate many disease defects with which school children suffer and to produce stronger and more capable children for the years with which the school has to deal. The problems of child care, feeding, health, nutrition, diseases, abnormalities, preventive hygiene and mental hygiene, and the dissemination among parents of proper ideas as to the care, nurture, training and instruction of their children are of such tremendous importance to the country and the State of South Australia.

The work done in this State in pre-school hygiene is not very extensive. The Kindergarten Training College of South Australia has supplied the following information:

There are nine free kindergartens in the State. Children enter at the age of two years and continue till six years old. There are approximately 450 children attending the nine kindergartens. These children are kept under medical inspection, but any treatment being undertaken by the children's own doctors or at public hospitals.

The Government Statist advised that the number of children in the State between the ages of two and five years inclusive is estimated at 50,000 approximately. About 4,000 of these have some opportunity of examination in connexion with the Education Department, and about 450 in the Free Kindergarten. Dental treatment is being carried out only in a limited way, mostly at the Adelaide Dental Hospital and the Adelaide Children's Hospital.

In the Minister of Education Report, 1929, he states there are 78,026 children at school between the ages of six and fourteen years. At private schools there are 14,000.

Quarantine.

This is a matter of Commonwealth administration in so far as vessels are concerned. There is, however, liaison with the State in the matter of infectious disease. The Commonwealth Department of Health notifies the State Health Department of all cases of disease present on board a vessel on arrival, gives addresses of any infected persons who have been permitted to land, and gives a list of passengers landing and their addresses. Persons with major quarantinable disease are landed at the Quarantine Station. Patients with minor disease, such as measles, diphtheria, scarlet fever *et cetera*, are sent to hospital or to their own homes, if suitable isolation can be arranged there. If suitable arrangements cannot be made to land them into hospital or to their own homes, they are removed to the Quarantine Station. The occurrence on board a vessel of a severer type of measles, diphtheria, scarlet fever *et cetera*, with heavy mortality, would be taken as an indication for removing infected persons to quarantine.

The Commonwealth Department of Health exercises control over all vessels, persons and goods arriving from oversea ports or engaged in interstate trading, and in respect of all animals and plants brought from any places outside Australia.

The Commonwealth possesses a quarantine station in each State for the purpose of human and animal quarantine. Torrens Island, near Port Adelaide, is used for that purpose.

Vessels from overseas, which have not *pratique* for South Australia, and desiring communication with the shore, must enter at a first port of entry. There are five first ports of entry in South Australia, namely, Port Adelaide, Port Lincoln, Port Pirie, Wallaroo and Thevenard.

A medical chief quarantine officer, with assistant quarantine officers, has been appointed in each State. This responsible officer is under the control of the Director-General of Health:

> For on his choice depends the safety
> and health of this whole State.
> (*Hamlet*, Act I, Scene 3.)

The *Quarantine Act* provides for the inspection of all vessels, including vessels navigated by air, from overseas, for the quarantine, isolation and disinfection of infected or suspected vessels, persons and goods, and, if thought necessary, the destruction of imported goods, animals and plants.

Quarantinable disease means smallpox, plague, cholera, yellow fever, typhus fever, leprosy or any disease declared by the Governor-General by proclamation to be a quarantinable disease.

Formerly every overseas vessel was examined in each State. Now those arriving from the east and the west are inspected at their first port of call, and if there are no cases of quarantinable disease or suspected cases on board, and the vessel is not less than eighteen days out from her last overseas port of call, *pratique* is granted for the whole of the Commonwealth.

The present freedom of Australia from certain diseases which are endemic in other parts of the world, justifies the Commonwealth in adopting precautionary measures not perhaps warranted in the already infected countries of the old world. The reliance which the State governments and the people

generally place in the *Quarantine Act* and its administration may be considered to be reflected in the relaxation of the vaccination regulations in all States; so that vaccination, except where there is an outbreak of smallpox, is carried out only by a small portion of the community.

The records of the Commonwealth Department of Health show how frequently vessels arrive from overseas with quarantinable disease on board and how seldom, relatively, there have been outbreaks of quarantinable disease in Australia.

Food and Drugs Act.

It is the duty of the Central Board of Health to enforce the *Food and Drugs Act* throughout the State. All local boards of health are constituted the local authorities for their respective districts, and it is their duty to put in force from time to time, as occasion may arise, the powers with which they are invested, so as to provide proper securities for the sale of food and drugs in a pure and genuine condition, and in particular to direct their officers to take samples for analysis.

The Central Board of Health acts as a supervisor and adopts an active policy of inspection and advice and also has concurrent jurisdiction with local boards. On the failure of a local authority to execute or enforce any of the provisions of the Act in relation to any article of food or drug, the Central Board may, by order, empower an officer to carry out such provisions and to recover the expenses incurred. Both the central and local authorities may appoint inspectors for the purposes of carrying the legislation into effect. Provision is made for the Government, on the recommendation of the Central Board of Health, to appoint persons possessing competent knowledge to be analysts. Every officer of health appointed under the *Health Act* has all the authorities and powers of an inspector appointed under the *Food and Drugs Act* in the district for which he has been appointed. The various districts in the metropolitan area are by the Act declared a county district, and a board, designated the Metropolitan County Board, is constituted for the purposes of the Act for that area. An advisory committee, composed of the Chairman of the Board of Health, the Professor of Chemistry of the University of Adelaide, the Government Analyst, Officer for Health for Adelaide, and three other persons conversant with trade requirements, is appointed.

Its function is to advise the fixing of standards and the making of regulations regarding articles of food and drugs for human consumption. The general objects of the Act are to secure the wholesomeness, cleanliness and freedom from contamination or adulteration of any food, drug or article, and the cleanliness of receptacles, places and vehicles used for their manufacture, storage or carriage. The sale of any article of food or any drug which is adulterated or falsely prescribed, is prohibited, as is also the mixing or selling of foods or drugs so as to be injurious to health. Power is given to any authorized officer to enter any place for the purpose of inspecting any article to be used as a food or drug, and to inspect articles being conveyed by road or rail. The officer may take samples for analysis or examination and may seize for destruction articles which are injurious for health or unwholesome. The regulation and restriction and conditions of the sale of poisons is provided for and the enforcement is performed by the Central Board of Health.

Special provision is made for the licensing of vendors of milk and the registration and inspection of their premises in order to secure wholesomeness, cleanliness and freedom from contamination of milk and the cleanliness of all receptacles, vehicles and vessels used in connexion therewith.

Review of a Few Matters of Interest that came within the Administration of the Central Board of Health in the State of South Australia, 1930.

Apart from the routine matters relating to all the activities that came within the administration of the Central Board of Health during 1930, a brief summary of subjects of interest is as follows.

Bacteriolytic or Septic Tanks.

In South Australia the Central Board of Health, as a result of experience extending over thirty years, has evolved a definite type of bacteriolytic tank which works automatically in respect to the treatment of the sewage and the disposal of the effluent.

Plans of all proposed tanks, with the connexions thereof, require to be submitted to and approved by the Central Board of Health, and an inspection of the installation is made by a trained officer of the Board and a permit issued by the Board before the tank is used.

Up to the present the Board has dealt with over 16,000 installations, and the individual plans of each of these are preserved in the Central Board of Health Office.

The premises at which tanks have been installed in cities, towns and country places throughout the State are varied as well as numerous. The list includes private houses, hotels, churches, church halls, boarding houses, restaurants, banks, business offices, hospitals (public and private), sanatoria, shops and stores for food and merchandise, manufacturing establishments, such as implement works, bottle works, timber mills; tramway shelters, railway stations, wheat-stacking sites, public conveniences on beaches, refreshment rooms above the sea at piers, tramway signal cabins, public pleasure grounds, camping grounds, racecourses, fire stations, sheep stations, schools and colleges, convents, public halls, theatres, mines, and all sorts of places where numbers of workers are employed or where people congregate.

Legislation in 1926 and 1927 gave power to municipal councils and district councils to require the installation of tanks in towns or townships. Certain of these authorities have exercised such

power, with the result that the various premises are provided with a system that is both satisfactory and without cost for working expenses, as the initial cost is the only charge.

Hospital Accommodation for Infectious Diseases.

Under the *Health Act* of 1898 power is given to local boards of health to enter into an agreement with hospital authorities for the reception and treatment of cases of infectious disease. In the metropolitan area all but one of the local boards have entered into such an agreement with the Adelaide Hospital, which is a Government institution. In country districts a fair number of local authorities have acted similarly with hospitals in or adjacent to their boundaries. In a number of cases local authorities have made no such agreement, but send patients into hospital and pay for their treatment. The great majority of local authorities in the metropolitan area and in the country endeavour to recover the amounts paid by them from the persons concerned. In cases where it is not possible for persons to pay, the amount is written off.

As regards the establishment of an infectious diseases hospital the following is taken from the City of Adelaide Year Book, 1931:

In 1921 a conference convened by the Adelaide Local Board of Health was held with representatives of the other local boards of health in the metropolitan area and of the South Australian Branch of the British Medical Association, to consider the question of hospital accommodation for infectious diseases.

As the result of this conference an arrangement was entered into under which the Government undertook to erect and equip a hospital for such cases, the local boards of health to be responsible for the maintenance and management. The Act to give effect to this arrangement was passed in 1922 and was brought into force by proclamation on 31st October, 1929. Under this a board called the Metropolitan Infectious Diseases Hospital Board is constituted as follows: The Lord Mayor (*ex officio*), chairman, one member appointed by each of the local boards of health for Adelaide, Port Adelaide and Unley, four by the other local boards, which for this purpose are arranged in four groups, two members by the British Medical Association.

The expenses and maintenance and management of the hospital are made by contributions from the local boards of health, such contributions to be based proportionately on the total assessed annual value of their districts. The expenditure so incurred by the local boards is chargeable against the health rate.

The hospital is declared to be a school of medical instruction in connexion with the University of Adelaide.

The buildings have been completed, but the hospital was not in commission.

Poliomyelitis Anterior Acuta.

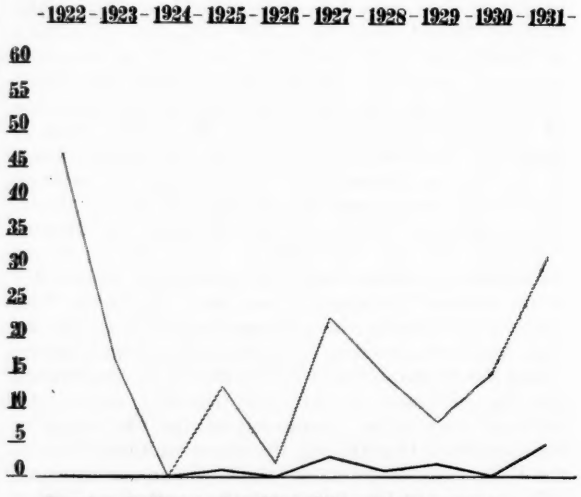
Poliomyelitis anterior acuta was declared by proclamation to be an "infectious" disease within the meaning of the *Health Act*, 1898, on February 23, 1922.

A committee to provide treatment for acute cases of anterior poliomyelitis by serum from convalescents was formed in September. It is composed of eighteen members, half of whom are nominated by the South Australian Branch of the British Medical Association and the remainder by the South Australian Division of the Australian Red Cross.

The committee has secured the cooperation of the Central Board of Health, which has furnished it with a list of the cases notified since 1922 and has arranged to supply particulars of future cases reported. From this information the committee will have knowledge of the distribution of cases and will be in a position to get into touch with possible donors.

The Central Board has drafter a *questionnaire* to be sent to officers of health of local boards in regard to cases that may be notified. In anticipation of a possible epidemic the committee purchased serum from Melbourne and Sydney, but has now completed arrangements for the preparation of serum locally. An honorary medical officer has been appointed for the first six months. About £200 has been raised for the purchase of equipment and the payment of donors. About eight patients in the early stages have been treated with successful results. At this juncture I should like to thank the Red Cross Society of this State for their good work to date.

I have prepared a graph comparing the notifications and deaths of poliomyelitis in South Australia and Victoria from 1922 up to the present date. They show the greater prevalence of the disease in our sister State.



Showing incidence of *poliomyelitis anterior acuta* in South Australia.

Pulmonary Tuberculosis.

The law gives local authorities certain powers of control over infected persons, infected articles and premises, and also in the matter of the protection of food. The statistics relating to notification of and deaths from this disease have shown improvement during recent years, as will be observed from the accompanying table (Table II).

Infant Welfare.

The City of Adelaide Municipal Year Book supplies the following information:

Table II.

Showing Deaths from Pulmonary Tuberculosis.

Year.	Notifications.	Deaths.
1921	517	337
1922	478	319
1923	478	334
1924	551	336
1925	540	332
1926	483	346
1927	467	335
1928	472	291
1929	458	302
1930	425	256

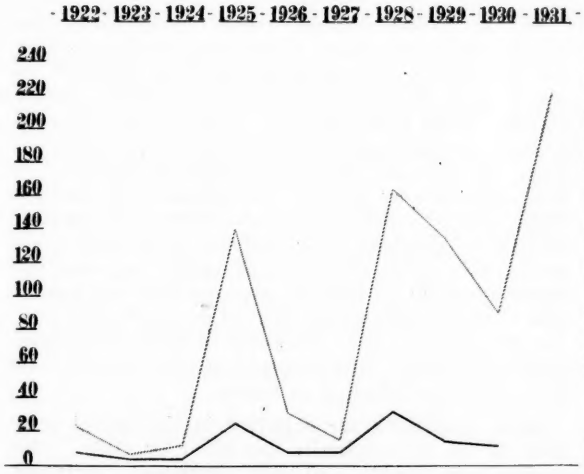
The Mothers and Babies' Health Association (which is subsidized by the Adelaide Council in its capacity as Local Board of Health for the City) was established to bring about a reduction in infant mortality and to build up a healthier and stronger race.

There are forty-five centres in Adelaide, suburbs and the country, and twenty fully trained nurses on the staff.

Lists of births throughout the State are received daily from the Central Board of Health. As, however, a considerable number of people fail to notify births, it is necessary to obtain the weekly lists of newly born babies from the Registrar-General of Births. In every district where the Association has established a baby health centre the infant mortality rate has decreased.

In twelve months ending 31st July, 1930, 70,000 visits were paid to the centres by mothers with babies, while the nurses made 38,000 visits to homes, and 4,479 books of advice were posted to mothers in outlying districts.

The Association is mainly supported by public subscription.



Showing incidence of *poliomyelitis anterior acuta* in Victoria.

Venereal Diseases Act, 1920.

The *Venereal Diseases Act*, 1920, is not yet in operation, but the provisions are to be carried out by the Inspector-General of Hospitals. The Minister administering the Act may arrange with any public hospital to provide free accommodation and treatment and may also establish hospitals and arrange for free examinations and free supply of drugs. Persons suffering from venereal disease are compelled to consult a medical practitioner or attend a hospital and place themselves under treatment.

No person other than a medical practitioner may attend or prescribe for patients.

Conclusion.

Before closing my address it is my duty to express to you my appreciation of the honour of occupying the position of your President during the year just terminated and to tender my most grateful thanks for all the help which I have received from members of the Council. Might I be permitted to acknowledge my indebtedness to the Honorary Medical Secretary of the Branch and also the Lay Secretary, both of whom have done yeoman service.

THE INFLUENCE OF INSULIN AND ADRENALIN ON CARBOHYDRATE METABOLISM.

By A. B. Corkill, M.B., B.S. (Melbourne),
Biochemist, The Alfred Hospital, Melbourne.
(From the Baker Medical Research Institute.)

Since their isolation, insulin and adrenalin have enabled us to add considerably to our knowledge of carbohydrate metabolism. You are all familiar with the marked influence that insulin has on this metabolism. Until recently, however, the rôle played by adrenalin has been rather neglected, and it is chiefly this aspect of the problem that I wish to discuss with you this evening.

In 1901 Blum noted that a subcutaneous injection of adrenalin provoked hyperglycæmia and, when administered in larger doses, glycosuria. The generally accepted explanation of this phenomenon was that adrenalin, acting through the sympathetic nerves to the liver, caused a breakdown of liver glycogen to glucose, which then escaped into the blood stream. Whilst not denying that the observed hyperglycæmia is dependent upon a mobilization of liver glycogen, yet I wish to present certain evidence in favour of an additional effect of adrenalin, namely, an extrahepatic action.

First, Cori has demonstrated that, even if all the glycogen present were mobilized, it would be insufficient to account for the degree of hyperglycæmia observed. A series of rats having fasted for twenty-four hours was injected with 0·02 milligramme of adrenalin per 100 grammes of body weight. An average hyperglycæmia of 174 milligrammes *per centum*, lasting for three hours, was produced. It was estimated that the liver glycogen before the administration of adrenalin was 218 milligrammes. Next, Cori attempted to determine how much artificially infused glucose would be necessary to produce in a normal rat a hyperglycæmia of similar magnitude to that observed after the injection of adrenalin. By means of a glucose infusion into the tail vein he found that, to produce a hyperglycæmia of 174 milligrammes *per centum* for three hours he had to infuse during that period 750 milligrammes of glucose. Thus, if the hyperglycæmia following adrenalin were due to a mobilization of liver glycogen, it would mean that

[1] Read at a meeting of the Alfred Hospital Clinical Society, February 24, 1931.

750 milligrammes were mobilized. Actually it was found that only 218 milligrammes were present before the adrenalin injection.

Again, if the only action of adrenalin were to mobilize liver glycogen to glucose, then it would logically follow that repeated injections should deplete the liver of glycogen. Some early experiments by Pollak and Kuriyama tended to show that actually the liver glycogen was increased in animals receiving adrenalin. At this time these results were apparently so contradictory that they were conveniently forgotten. However, recently a number of careful investigations have clearly demonstrated the validity of these early results. In some experiments animals were deprived of food and were given strychnine, which is known to deplete the liver of glycogen. Adrenalin was then injected and it was found that more liver glycogen was present in these animals than in those merely fasting. In most experiments of this type the animals were killed from eighteen to forty-eight hours after the adrenalin injection.

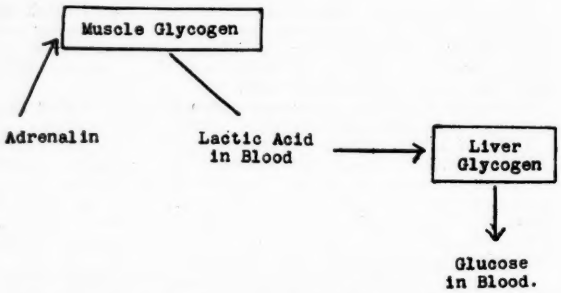
Whilst working on the problem of insulin and adrenalin relationships, I was able to show that in young rabbits that had fasted during twenty-four hours, a suitable dose of adrenalin was able to lay down liver glycogen after an interval of two hours. In the following experiment young rabbits from six to eight weeks old that had fasted for twenty-four hours, were used. Some were used as controls and the others were injected with adrenalin and killed two hours later. The figures clearly demonstrate that under the influence of adrenalin a marked deposition of liver glycogen has occurred (see Table I).

Table I.

Controls.		After Injection of Adrenalin (0·02 milligramme per 100 grammes body weight).	
Liver Glycogen Percentages.	Blood Sugar Percentages.	Liver Glycogen Percentages.	Blood Sugar Percentages.
0·45	0·135	1·87	0·168
0·46	0·141	0·92	0·215
0·46	0·145	1·60	0·155
0·10	0·138	1·94	0·163
0·13	0·148	1·20	0·148
0·10	0·120	1·40	0·163
Average. 0·28		Average. 1·49	

Clearly, then, in view of the foregoing, a simple mobilization of liver glycogen cannot explain all the observed facts.

Cori has recently carried out a series of extensive experiments on rats receiving adrenalin from which he established carbohydrate balances. He was able to show that in all cases liver glycogen increased, but that muscle glycogen decreased; and he conceived the idea that in some way the muscle glycogen had been transported to the liver. At first sight the explanation might appear to be simple, namely, that the hyperglycæmia represents the breaking down of muscle glycogen to glucose and its direct transport to the liver. Unfortunately this cannot be. Mann and Magath have shown that muscle glycogen cannot directly contribute to the circulating glucose. Thus in the dog whose liver has been removed, the blood sugar was found to fall steadily, and injections of glucose were necessary to maintain the normal sugar level in the blood. Again, as Claude Bernard originally noted, there is a fundamental difference between the behaviour of liver and muscle glycogen. The former breaks down to glucose and the latter to lactic acid. Correlating these facts, Cori conceived a cycle as follows:



Under the influence of adrenalin muscle glycogen is transformed into lactic acid, which is conveyed in the blood stream to the liver, where it is reconverted to glycogen and ultimately to glucose.

In the intact animal, where most likely the several suggested stages towards the ultimate effect are simultaneously in progress, it is difficult to obtain more than qualitative data in support of this theory. Thus Cori was able to detect an increase in liver glycogen and a decrease in muscle glycogen. Also, he was able to demonstrate an increase in liver glycogen by feeding the animal with dextrolactic acid. In order to obtain evidence in support of Cori's hypothesis, it is necessary to gain some quantitative idea of the relationship between the breaking down of muscle glycogen and the formation of lactic acid under the influence of adrenalin. For this purpose it is necessary to devise simplified conditions under which this suggested change can be isolated and studied at leisure.

Such conditions are supplied in the spinal eviscerated cat, as originally described by Dale. The brain and spinal cord are destroyed, artificial respiration is then initiated, and after ligating the appropriate blood vessels the abdominal viscera are removed. The liver is left *in situ*, but has its blood supply cut off. It forms a cul-de-sac over the inferior *vena cava*, and though excluded from any active participation in the general circulation, yet to some slight extent glucose resulting from glycogen breakdown can leak into the general circulation. To all intents and purposes the eviscerated cat is simply a muscle preparation. It is found necessary to supply glucose, since, if left alone, the blood sugar rapidly falls. By means of a suitable sugar infusion (Dale and Burn) it is possible to maintain the blood sugar constant at any desired level.

If in such a preparation adrenalin causes a breakdown of muscle glycogen, it should be possible to study the immediate products of this breakdown.

In performing an experiment of this nature the usual procedure was to arrange the glucose infusion at a predetermined rate, and, when the blood sugar had attained a constant level, to take samples of muscle for glycogen determination from one hind limb and then, two hours later, similar samples from the other hind limb. Dale *et alii* have shown that although different muscles have varying glycogen content, yet corresponding muscles of both hind limbs have, within the experimental error, the same glycogen content. The following experiment will illustrate this:

A 6% glucose solution was infused at the rate of 432 milligrammes per hour into a cat weighing 3·8 kilograms. Initial muscle samples were taken when the blood sugar had attained a steady level, and final samples two hours later. The results are shown in Table II.

Table II.

Muscles.	Initial Glycogen Percentages.	Final Glycogen Percentages.
Tibialis anticus ...	0·46	0·43
Gastrocnemius ...	0·46	0·45
Quadriceps femoris	0·45	0·44
Sartorious	0·27	0·11
Vastus internus ..	0·48	0·57
Average ..	0·42	0·41

Having ascertained that we could reproduce control experiments of the type shown above, we then decided to investigate the effect of adrenalin in this special preparation. At the outset it became apparent that a marked decrease in muscle glycogen took place. A typical experiment is shown below:

Glucose was infused at the rate of 240 milligrammes per hour into a cat weighing 3·25 kilograms. When the blood sugar had reached a constant level, initial muscle samples were taken. Infusion of a one in 10,000 solution of adrenalin was then commenced and continued for two hours, at the end of which time the final muscle samples were taken. The result is shown in Table III.

Table III.

Muscles.	Glycogen Percentage before Infusion of Adrenalin.	Glycogen Percentage after Infusion of Adrenalin.	Difference.
Tibialis	0·79	0·57	− 0·22
Gastrocnemius	0·85	0·60	− 0·25
Sartorius	0·76	0·47	− 0·29
Quadriceps femoris	0·72	0·49	− 0·23
Vastus	1·14	0·74	− 0·40
Average ..	0·85	0·57	− 0·28

The next step was to investigate quantitatively changes occurring in the lactic acid of the blood and muscles. In order to view these changes in their true perspective it was necessary to know their variations, if any, in an experiment in which adrenalin was not administered. For this purpose all the known factors were taken into account and an attempt was made to draw up a balance sheet. The factors concerned were changes in muscle glycogen, in muscle sugar and lactic acid, in blood glucose and lactic acid. By means of the Schuster respirometer we were able to measure the oxygen consumption, and since we believed the respiratory quotient of this preparation to be unity, we could directly convert the oxygen consumed into oxidized glucose. A balance sheet from a control experiment is shown below.

Table II, showing the constancy of hind limb glycogen, is really taken from this experiment; there is actually a slight loss, which is, however, quite insignificant when compared to the loss observed following administration of adrenalin (see Table III).

Balance Sheet from a Control Experiment.

	Grammes.		Grammes.
Glucose infused	0·86	Glucose equivalent of oxygen used	2·04
Fall in muscle glycogen	0·19		
Fall in muscle sugar	0·19		
Fall in muscle lactic acid	0·57		
Fall in blood sugar ..	0·03		
Fall in blood lactic acid	0·11		
Glucose from liver ...	0·06		
Total carbohydrate disappearing	2·01	Carbohydrate accounted for	2·04

It will be observed that a very close balance has been arrived at. In a series of similar experiments the largest discrepancy observed was 0·3 to 0·4 gramme. It is interesting to note that the muscle and blood lactic acid actually decreased.

Table III is taken from an adrenalin experiment, the balance sheet for which is as follows:

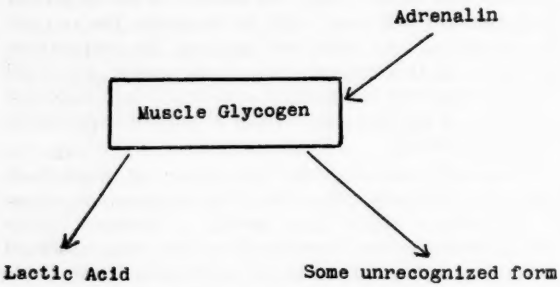
Balance Sheet for Adrenalin Experiment.

	Grammes.		Grammes.
Glucose infused	0·74	Glucose equivalent of oxygen used	2·09
Fall in muscle glycogen	4·45	Rise in muscle lactic acid	0·59
Fall in blood sugar ..	0·06	Rise in muscle sugar	0·48
Glucose from liver ..	0·18	Rise in blood lactic acid	0·12
Total carbohydrate disappearing	5·43	Carbohydrate accounted for	3·28

In this instance 2·15 grammes of carbohydrate were not accounted for.

Contrasting this with the control experiment, it will be observed that, whereas in the latter the fall in muscle glycogen was 0·19 gramme, in the former a fall of 4·45 grammes was noted. This was unlike the control experiment in that a balance was not obtained. In a series of similar experiments the same discrepancies were found. It would appear from these results that under the influence of adrenalin muscle glycogen is broken down partly to lactic acid and partly to some form that could not

be estimated with the chemical methods used by us. Thus:



Leaving for the moment the adrenalin question, let us consider the action of insulin. Dale and others who studied this problem also in the eviscerated spinal cat, were able to show that in this preparation insulin produced its typical effect on the blood sugar. They were also able to demonstrate that the greater fraction of the glucose which disappeared was stored as muscle glycogen, and the remainder was oxidized. These experiments, unlike the adrenalin experiments just discussed, gave a complete balance, so that all the sugar which disappeared could be accounted for. It is thus seen that insulin and adrenalin have directly opposite effects on muscle glycogen, the former removing glucose from the blood stream and storing it in the muscles, whilst the latter breaks it down to lactic acid.

From the clinical standpoint adrenalin is capable of relieving an insulin hypoglycæmia. So far, however, there is no evidence of any direct antagonism between insulin and adrenalin. Accordingly, we decided to test this possibility. A spinal eviscerated preparation was set up and a small dose of insulin (five units), which produced a moderate fall in the blood sugar, was injected (see Table IV). A similar

TABLE IV.

A 4% solution of glucose infused at a rate of 9·6 cubic centimetres per hour into a cat weighing 3·7 kilograms.

Time.	Blood Sugar Percentage.
2.0	0·332
2.20	0·332
2.40	0·304
3.0	0·304
Five Units of Insulin Injected.	
3.25	0·304
3.45	0·240
4.10	0·222
4.30	0·222

preparation was then set up, and when the blood sugar had attained a steady level, an infusion of a one in 10,000 solution of adrenalin was commenced and continued throughout the experiment. After the adrenalin had been in action for sixty-five minutes, five units of insulin were injected, with the result shown in Table V.

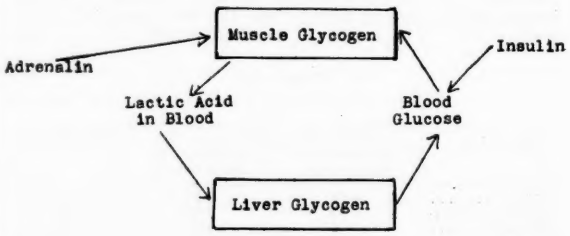
TABLE V.

A 4% solution of glucose infused at a rate of 9·6 cubic centimetres per hour into a cat weighing 3·3 kilograms.

Time.	Blood Sugar Percentage.
12.45	0·280
Adrenalin Infusion Commenced.	
1.10	0·280
1.30	0·294
1.50	0·300
Five Units of Insulin Injected.	
2.15	0·264
2.30	0·253
2.50	0·240
3.10	0·219

It cannot be suggested that the adrenalin infusion has in any way inhibited the action of the insulin. In another experiment of this type samples of muscle glycogen were taken and it was found that although insulin produced its typical effect on the blood sugar, yet on making out a balance the muscles were found actually to have lost in glycogen content. Now, insulin alone in these circumstances would store the disappearing blood glucose as muscle glycogen, so that the loss in muscle glycogen would represent excess of discharge over storage.

It would appear that insulin and adrenalin, when simultaneously present, produce a combined effect of just the type which could be predicted from their separate actions: (i) under the action of insulin a removal of glucose from the circulation by its deposition as muscle glycogen, and (ii) under the action of adrenalin, a loss from the muscles of glycogen which partly reappears as lactic acid and is to some extent lost to the ordinary methods of analysis. Part of this glycogen removed by adrenalin would, according to Cori, be reconverted into liver glycogen, so that apparently we have a cycle somewhat as follows:



Insulin.

Since the isolation of insulin a puzzling discrepancy has been noted between the actions of insulin on the diabetic and on the normal animal. In the diabetic animal it readily renews the store of glycogen in the depleted liver. On the other hand, there is a considerable amount of evidence to show that in some species of normal animals insulin, when injected, actually diminishes the amount of glycogen in the liver. The most reasonable explanation of this paradox seems to be that offered by Dale *et alii*. The two cardinal defects in diabetes are:

(i) inadequate use and storage of carbohydrate, and (ii) excessive wasteful production of carbohydrate from proteins and fats. Insulin in appropriate dosage remedies these defects. It is, therefore, reasonable to assume that insulin in excess (and it is in excess when it is injected into a normal animal) will produce each of its normal effects to an abnormal degree. In other words, we should expect it (i) to accelerate the storage of glucose as glycogen to a rate above normal, and (ii) to inhibit the formation of carbohydrate from proteins and fats to a rate much below normal.

1. Dale *et alii*, working, as already mentioned, with the eviscerated spinal cat, were able to show that under these conditions excess of insulin undoubtedly accelerated the rate of glycogen storage in the muscles.

2. Insulin, as Dudley and Marrion originally showed, causes marked depletion of carbohydrate reserves in the mouse. In such an animal the amount of available carbohydrate at any moment is only sufficient for about twenty minutes' metabolism. Hence the normal metabolism can only be maintained by the rapid new formation of carbohydrate in the liver. If this new formation be stopped, then the existing supply of carbohydrate will be exhausted in about twenty minutes. The results which follow the injection of insulin into a mouse are exactly those which, under such conditions, we should expect. There is an initial sharp rise in the respiratory quotient, indicating that during a brief period the oxidation is concentrated on preexisting carbohydrate. It is also probable that for a short period glucose is removed from the circulation and stored as glycogen. As soon, however, as the existing carbohydrate is used, and this occurs in about twenty minutes, the animal should have convulsions with its initial stores of carbohydrate completely exhausted. This is what actually occurs.

It has been stated that a considerable amount of evidence tended to demonstrate a decrease in liver glycogen following the administration of insulin to normal animals. This effect is clearly defined in such an animal as the mouse, and I have outlined the influence of inhibition of new carbohydrate formation from proteins and fats on the causation of a decrease in liver glycogen. Other workers have studied the effects of insulin in normal rabbits, but the results obtained have been very contradictory. Some have claimed that insulin causes a deposition of liver glycogen and others have denied this. In experiments of this nature the usual procedure was to take two groups of animals, either fed or fasting for twenty-four hours. One group was used for control purposes and the livers were analysed for glycogen content; the resulting figures were compared with those obtained from the other group, the members of which received insulin. Macleod has pointed out that there is a considerable chance of error in experiments of this type, chiefly "owing to the fact that one can never predict in (adult) rabbits the percentage of glycogen in the liver." Recently, however, Goldblatt has overcome this disadvantage. He found that in young rabbits from six to eight weeks old which had fasted during twenty-four hours, the amount of liver glycogen was small and remarkably constant. Insulin (0·5 unit) administered to such a young rabbit caused a deposition of liver glycogen. I was able to confirm these results and also to obtain some rather suggestive figures for the muscle glycogen of these young rabbits. The figures in Tables VI and VII are taken from a number of experiments which, since they were performed under identical conditions, may be conveniently grouped together.

TABLE VI.
Showing observations in normal controls.

Weight of Rabbit in Grammes.	Liver Glycogen Percentage.	Muscle Glycogen Percentage.
476	0·24	0·19
480	0·25	0·18
462	0·60	0·21
464	0·20	0·30
852	0·28	—
582	0·08	0·66
800	0·12	0·42
690	0·87	0·08
904	0·42	0·12
804	0·33	0·30
676	0·22	0·27
Average. 654	0·30	0·27

TABLE VII.
Showing observation after injection of 0·5 unit of insulin.

Weight of Rabbit in Grammes.	Liver Glycogen Percentage.	Muscle Glycogen Percentage.
583	3·72	0·15
581	3·66	0·13
407	2·10	Trace
520	3·20	0·07
645	1·20	0·27
715	2·73	0·24
860	2·03	0·19
672	1·90	0·04
521	1·47	0·08
748	1·33	0·20
598	1·00	0·12
Average. 620	2·22	0·14

It will be observed that whereas the liver glycogen of the control animals averages 0·30%, that of the insulinized animals is markedly increased, namely, 2·22%. It will also be noted that there is a significant decrease in the muscle glycogen of these latter animals. Now this is precisely what Cori found when he injected small doses of adrenalin into rats. I have already discussed the significance of these results, namely, that under the action of adrenalin muscle glycogen is split into lactic acid, conveyed by the blood stream to the liver, where it is reconverted to glycogen, and ultimately to glucose. You will also remember that I was able to demonstrate that small doses of adrenalin in young rabbits caused a marked deposition of liver glycogen (see Table I). (In addition, the muscle glycogen in the rabbits receiving adrenalin was decreased.) Apparently, then, insulin and adrenalin have the same effect when administered to young

rabbits, namely, a deposition of liver glycogen with a simultaneous depletion of muscle glycogen. What, then, is the connexion between these phenomena?

In a sense the deposition of glycogen in the livers of young rabbits receiving insulin injections is just as puzzling as the diminution of glycogen found in mice. Even if all the circulating glucose were removed and stored in the liver, there would not be sufficient to account for the observed gain.

Goldblatt has suggested that in the young rabbit insulin acts by locking up as glycogen in the liver the glucose derived from the alimentary canal. This explanation is rather difficult to accept in view of the fact that a deposition of glycogen occurs even if these young rabbits are starved for several days, the alimentary canal thus being rendered practically free from food. Consequently we are driven to believe that the liver derives glycogen from some source within the body.

A possible explanation to the puzzle is afforded by the work of Cannon, McIver and Bliss, who call attention to the fact that the hypoglycæmic symptoms produced by insulin, namely, pallor, rapid pulse, dilatation of pupils and profuse sweating, are indications of sympathetic activity. To support their hypothesis that insulin hypoglycæmia leads to a secretion of adrenalin, these authors conducted a series of experiments in which the cardiac rate was observed following the injection of insulin. It was found that following the injection the blood sugar fell to a level at which the beating of the denervated heart began to be accelerated. If the suprarenals were previously removed, a fall in the blood sugar did not produce a cardiac acceleration. These authors concluded that the cardiac acceleration was directly due to an increased adrenal discharge. Now these observations have a very important bearing on the problem just discussed.

Briefly recapitulating the facts, we find that insulin administered to young rabbits causes a deposition of liver glycogen and depletion of muscle glycogen. Adrenalin can produce exactly identical results. Next we have the work of Cannon, McIver and Bliss, which shows that insulin hypoglycæmia leads to an accelerated output of adrenalin. In view of these facts it is tempting to suggest that the deposition of liver glycogen following the administration of insulin in young rabbits is not primarily due to a direct action of insulin, but to a secondary output of adrenalin. This theory would have the advantage that the known facts can all be explained in physiological terms. Such a state of affairs would also fit in very well with the cycle of carbohydrate metabolism which is shown in the first diagram (cycle).

At present we have no direct evidence of the exact cycle of events that occurs in the human body, but it is extremely probable that a relationship similar to that already described as existing between insulin and adrenalin also exists here.

There are several other important hormones, such as those of the thyreoid and the pituitary, which I have not discussed. Both these agents also exert powerful influences on carbohydrate metabolism. The hormone from the posterior lobe of the pituitary gland can be separated into two fractions, an oxytocic fraction and the vasopressor fraction. The latter fraction is of extreme interest from the standpoint of carbohydrate metabolism. By itself it causes no definite changes in the blood sugar, yet it is capable of rapidly abolishing an insulin hypoglycæmia. In this respect it is far more potent than adrenalin and it is rather surprising that it is not used more frequently to combat insulin hypoglycæmia. Even when a well-developed hypoglycæmia is present, pituitary extract is capable of rapidly restoring the blood sugar to its normal level. There is another important, even if somewhat paradoxical, action of pituitary extract. Not only is it capable of relieving insulin hypoglycæmia, but it can also prevent the hyperglycæmia produced by adrenalin. At present the explanation of this phenomenon is not clear. A few preliminary investigations which were carried out by Marks and myself in Dale's laboratory, showed that this peculiar action of pituitary extract is probably hepatic and not peripheral.

Bibliography.

The work discussed in this paper is embodied in the following papers:

"The Effect of Adrenaline on Muscle Glycogen," by A. B. Corkill and H. P. Marks, *Journal of Physiology*, Volume LXX, page 67.

"The Influence of Insulin on the Distribution of Glycogen in Normal Animals," by A. B. Corkill, *Biochemical Journal*, Volume XXIV, page 779.

THE TREATMENT OF SNAKE-BITE IN AUSTRALIA.

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Success in the treatment of bites by venomous snakes depends in large measure upon the immediate application of first aid. It is important to be able to recognize at the outset whether the bite is by a venomous or a non-venomous species. The bite of a non-venomous snake need not cause any concern, apart from the possibility of the development of septic infection in the wound. This may be guarded against by thorough cleansing with soap and water, followed by the free use of an antiseptic—weak cyllin, carbolic acid or Condy's fluid—and the application of an antiseptic dressing.

Character of Puncture Marks from Teeth.

The bites of non-venomous snakes usually show many puncture marks, four rows being made by the numerous and well developed maxillary and palatine teeth.

In bites by venomous snakes the puncture marks may vary from one to four; one if only one fang

has entered, two if no reserve fangs are present, and three or four of there is a reserve fang on one or both sides.

In a bite by a venomous snake, the distance of the single punctures apart (if there are no reserve fangs) or the distance of the pairs of puncture marks (if reserve fangs are present) affords some indication of the size of the snake and of the probable quantity of venom injected.

Bites by the rarer venomous snakes of small size (about a foot or eighteen inches long, and not exceeding the thickness of a finger) are, for the most part, not dangerous to man. In regard to the young of the common venomous species, bites from reptiles of this size may prove fatal, especially in children. Energetic treatment should therefore be adopted in cases of bites by these snakes.

It is important, from the standpoint of antivenene treatment, to be able to recognize at least the common venomous snakes. These, in order of their danger to man, are: (i) the death adder, *Acanthophis antarcticus*; (ii) the tiger snake, *Notechis scutatus*; (iii) the copperhead, *Denisonia superba*; (iv) the common brown snake, *Demansia textilis*; (v) the red-bellied black snake, *Pseudechis porphyriacus*.

The mortality following the bites of these species is about 50% for the death adder, 40% for the tiger snake, and less than 10% for the brown snake. It is doubtful if the bite of the black snake is ever fatal for an adult, and the very few recorded deaths have possibly resulted from copperhead bites. Bites by the copperhead are rare, although this snake is a very common one.

Though it is not possible in the short space available here to give sufficient data for the certain identification of even the commoner venomous snakes, we have inserted a few notes on their most striking characteristics.

The death adder is a snake with a short, stout body. It is brown, red or grey in colour, with darker cross bands; it has a broad, rough-scaled head and a yellow spine at the tip of the tail, which tapers off very sharply. It is nocturnal in habit, buries itself in sand or leaves in the day time, and generally does not strike till touched. Its length is from eight or nine inches to two feet six inches. It occurs in Queensland, New South Wales, South Australia and Western Australia.

The tiger snake is a much larger species. It is broad-headed, and average specimens are from three to five feet in length; it shows great variation in colour and, except certain black and dark brown forms occurring in the islands of Bass Straits and Tasmania, is transversely banded. The colour varies from light grey to dark green or orange with dark transverse bands. This species is almost unmistakable. It occurs in every State in Australia.

The copperhead is a sluggish snake met with principally in swampy country. It is shorter and stouter than the brown snake, but is likely to be confused with the red-bellied black snake, for it shows great variation in colour. Specimens may be yellow, brown or black, the belly is usually yellowish. In many specimens orange or red lateral scales give the appearance of a longitudinal band of that colour down each side of the reptile. The average length is from three to four feet. It occurs plentifully in New South Wales, Victoria and Tasmania and near the Victorian border in South Australia.

The common brown snake is a longer and more slender reptile, whose length ranges up to six feet. It has a narrower head and is brown or grey in colour. Apart from its general build and great speed in the field, it may readily be distinguished from the copperhead by its divided anal scale and paired subcaudal scales. It strikes higher from the ground than the other Australian species. It is widely distributed throughout Australia.

The red-bellied black snake is a large, solid snake of a black colour with a metallic sheen. The lateral scales of the body and the belly and ventral scales are red. It is found in swamps and on hillsides, near river flats or swampy ground. Like the last mentioned snake and unlike the copperhead, the anal scale is divided and some of the subcaudals are paired. It occurs in Queensland, New South Wales, Victoria and South Australia.

First Aid Treatment.

First aid treatment varies with the site of the bite. If the bitten part be a limb, a ligature or ligatures should be applied within a few seconds after the bite. If the bite be upon a part where ligature is impossible, excision of a small area surrounding the fang punctures should be carried out without delay.

Ligature.

A ligature is of value only in retarding, while it is applied, the entry of venom into the circulating blood. In the instance of bites by brown snakes (*Demansia textilis*), the venom of which contains a powerful and diffusible coagulant, the ligature may prevent death by checking thrombosis. The ligature should be applied immediately, for the neurotoxins of all Australian snake venoms are rapidly diffusible.

The ligature should be sufficiently firmly applied to obstruct the arterial flow into the ligated part. It should be placed round a part of a limb containing only a single bone. Ligatures round the wrist, forearm, ankle and calf are valueless, since deeply seated vessels lying between two bones cannot be obstructed. Under these circumstances the ligated part becomes very swollen and the venom leaks steadily into the circulation while the ligature is still in position. In every case of a bite upon a limb a ligature must be applied upon either the thigh or the arm above the elbow joint.

The properly applied ligature gives time for local treatment to be carried out and slows down the entry of venom during the time which elapses before antivenene can be administered.

The ligature may be left in position for half an hour, after which time it should be lifted for at

least a minute or until the ligated part becomes pink with the entry of blood, then the ligature is reapplied at once. This procedure should be carried out every quarter of an hour to avoid serious damage to the tissues by prolonged local anoxæmia. Under these conditions a ligature may be safely used for one to two hours, when it is probably wise to discard it altogether.

In bites by tiger snakes the ligature should be removed as soon as antivenene has been injected, but if antivenene is not available early, it should be given as soon as possible after the ligature has been removed.

Excision.

Excision should be carried out immediately after a bite upon an area where a ligature cannot be applied. N. Hamilton Fairley advises excision of the skin and subcutaneous tissue over an area, including the punctures, a little greater than a square inch. The excision should be extensive enough to include the deepest point reached by the fangs.

Excision is of no value whatever unless carried out at once and is therefore seldom made. The surface should, if possible, be washed or licked to remove venom on the surface of the skin before excision. It is of value to apply suction either by the mouth or by a dry cup immediately afterwards. The surface of the excision and the surrounding skin may with advantage be washed with weak potassium permanganate solution if this substance is available.

If the bite be upon an area above which ligatures can be applied, excision of the area including the fang marks is still the best treatment if applied immediately.

Incision.

Incision is not a very satisfactory method. Theoretically the incisions should be made along the tracks of the fang punctures, starting from the punctures and being roughly at right angles to a line joining them. As the tracks are directed somewhat obliquely backwards and inwards, and it is often uncertain exactly how the snake gripped the part, it is better to make two nearly parallel incisions through the region of each puncture, starting in front of the line joining the marks and extending behind it. Blood should be squeezed through the incisions out of the ligated part by bandaging it firmly from the ligature downwards. Persons applying first aid to themselves will frequently use this method, as it requires great physical courage to perform excision.

Local Venesection.

Local venesection may be carried out in snake-bites on the limbs if the patient is seen by a medical man within the first hour or two and the ligature has not yet been removed. In principle it depends upon washing out the part with the patient's own blood. A second ligature, theoretically tight enough to obstruct the venous return, but not the arterial flow into the limb, is placed in position immediately distal to the arterial ligature. An incision is made into one of the veins draining the area in which the bite is situated, and a succession of small blood lettings from this vein are carried out by lifting the arterial ligature for a minute or two and leaving the venous ligature in position. Some venom (in animal experiments about a third to a half of the amount injected) can be removed in this way, and the treatment is useful if antivenene is not available. In a child the blood can be replaced by transfusion from a suitable donor. In an adult a pint or a pint and a half of blood can be removed in flushing the bitten part in this way.

Antivenene.

Antivenene is as yet available for use only against the bites of the tiger snake (*Notechis scutatus*) and is not of practical use against the venoms of other species. It is put up in ampoules containing approximately 30 cubic centimetres, or 1,500 units, an amount sufficient to neutralize 15 milligrammes of dry tiger snake venom *in vitro*. It should be administered intravenously in a dose of 3,000 units. More should be given if further symptoms develop after its administration. Intravenous injection is of value because, as Martin pointed out, it rapidly neutralizes venom which is already circulating in the blood. The earlier the antivenene can be administered, the better the chances of recovery. Even to those patients who are brought for treatment late, that is, more than twelve hours after being bitten, large doses of antivenene should be given. In a recent case of tiger snake bite the patient was admitted with serious symptoms twenty-four hours after being bitten; the administration of 4,500 units of antivenene intravenously and a further 3,000 units intramuscularly (to maintain a high concentration of antivenene in the blood) was followed by a dramatic recovery.

Therefore, one should not despair of any victim of tiger snake bite, but should begin treatment with antivenene as soon as possible.

The subcutaneous administration of antivenene is of little value, since the absorption rate of serum is so much slower than that of the rapidly diffusible snake poisons.

A number of persons are hypersensitive to horse serum, and in such cases rapid intravenous injections of serum may cause acute anaphylactic shock. There may or may not be a history to guide one of attacks of asthma or hay fever, or of having received previous injections of serum. Horse asthmatics are particularly prone to react dangerously following the injection of horse serum. It is much safer, therefore, to test the patient before giving an intravenous dose by injecting intradermally 0·1 cubic centimetre of diluted horse serum. This may be prepared rapidly by adding one part of serum to nine parts of sterile normal saline solution, mixing well, and injecting one-tenth of a cubic centimetre of the diluted serum into the skin.

A control injection is made of 0·1 cubic centimetre of normal saline solution prior to giving the

diluted serum. Wheals are produced at the site of injection in both cases; that produced by the salt solution disappears in the course of a few minutes. If the patient is not hypersensitive, the wheal produced by the serum fades almost as quickly. If a positive result to the test is obtained, a genuine urticarial wheal appears which may increase in size and be surrounded by an area of erythema. Itching frequently accompanies a positive reaction.

It should be possible to determine within twenty minutes of the injection what type of reaction the patient will give.

Should the patient prove hypersensitive to serum, it is dangerous to give serum intravenously without first desensitizing the patient. To do this, an initial dose of 0·025 cubic centimetre is given subcutaneously. If noticeable symptoms due to serum do not appear, half an hour later give 0·1 cubic centimetre of the serum subcutaneously and await results. If appearances are still favourable, then in half an hour give 1·0 cubic centimetre of serum subcutaneously. This is a much more rapid method of desensitizing than that usually recommended, of commencing with 0·025 cubic centimetre and doubling the dose at half-hourly intervals until 1·0 cubic centimetre has been given subcutaneously.

However, it must be remembered that it is necessary to act quickly in giving serum in a case of snake-bite.

Provided that no symptoms develop as a result of giving the third dose of serum, it is justifiable to proceed with the serum treatment without further delay.

As an initial intravenous injection give 0·1 cubic centimetre of serum. If no symptoms develop, proceed very carefully with the remainder of the serum. It should be given very slowly, and if unfavourable symptoms develop, administration should be interrupted for the time being.

For the prevention of serum anaphylactic shock in man, one may give 1·3 milligrammes (one-fiftieth of a grain) of atropine sulphate by subcutaneous injection about half an hour before giving serum intravenously.

For the treatment of anaphylactic shock, adrenalin chloride, in a dose of 0·5 to 1·0 cubic centimetre of one in 1,000 dilution should be kept in readiness and given if necessary.

Should the case be desperate and time of the utmost importance, the possible risk of causing serious anaphylactic symptoms should be disregarded and energetic treatment by intravenous and intramuscular injection should be begun without delay.

General Treatment.

The patient should be made to rest. He should not walk about, for the increased circulatory rate induced by exercise will speed up absorption of the poison. Furthermore, rest is desirable on general grounds, and no additional stress should be inflicted on the nervous system.

Alcohol is undesirable except for those accustomed to its use, and even then should be given only in small amounts. The best beverage as a stimulant is hot black coffee. Strychnine is of doubtful utility and in large and repeated doses may be dangerous to the patient. Pituitrin is possibly of value.

Circulatory shock should be combated by keeping the patient warm and giving plenty of fluid.

The supreme danger is that of respiratory failure, and the use of morphine is therefore contraindicated.

Finally, it is of the first importance that the patient should not be allowed to become the victim of his own fears.

The great majority of snake-bitten persons should recover if adequate treatment is adopted. In cases of bites by the tiger snake, the mortality, if anti venene is used, should be negligible. Otherwise the death adder is the only snake the bite of which is still likely to be attended by a substantial mortality. Treatment of bites by this snake should be energetic. If the patient be seen immediately after the bite, excision of the bitten area should be performed. If the first few minutes have passed, but the patient be seen within two hours of the bite, treatment by local venesection should, if possible, be performed, provided that a ligature has been previously applied.

Reviews.

DIABETES MELLITUS.

Our knowledge of the treatment of *diabetes mellitus* has grown enormously in the past decade; there is a danger that we may become dazzled by our therapeutic successes and forget how numerous and how important are the ætiological, physiological and pathological problems which this baffling disease still presents. Dr. Shields Warren, in his monograph, "The Pathology of Diabetes Mellitus," places the pathological situation before us with extreme clarity and with a refreshing lack of that prejudice and bias which have tended to cloud the views of so many workers in the field of diabetic pathology.[1]

Dr. Warren has two outstanding qualifications for his task; he has been trained by Dr. F. B. Mallory and he has worked in close conjunction with the diabetic clinic controlled by Dr. E. P. Joslin, from which has emanated such a vast amount of diabetic wisdom. The monograph is chiefly based upon a series of three hundred autopsies personally carried out by the author on cases of diabetes. The findings have been carefully correlated with the work of earlier investigators, and in the result we have a clear and unbiased presentation of all that is known of the morbid anatomy of diabetes at the present time.

Particular care has been taken to provide adequate controls for all observations on diabetic tissues. As Dr. Warren himself states: "So much attention has been paid to even minor lesions in the pancreases of diabetics, without much regard to changes seen in the pancreases of non-diabetics, that a tendency has developed to relate complacently almost any type of pancreatic lesion with diabetes." The ætiological importance of lesions affecting respectively the whole pancreas and the island tissue alone is carefully assessed; each of the typical lesions is described in detail and the descriptions are greatly aided by the excellent microphotographs with which the book is provided.

[1] "The Pathology of Diabetes Mellitus," by Shields Warren, M.D., with a Foreword by E. P. Joslin, M.D.; 1930. London: Baillière, Tindall and Cox. Royal 8vo., pp. 223, with 33 engravings and two coloured plates. Price: 35s. net.

That diabetic pathology concerns the clinician at least as much as the pathologist, inasmuch as such a large proportion of it is preventible, is very clearly shown by the chapter on the relationship between diabetes and arteriosclerosis, a subject which is giving so much food for thought to physicians at the present time. Dr. Warren hopes—and gives reasonable grounds for his hope—that with the gradual improvement in therapeutic technique the incidence of this distressing and frequently fatal complication will gradually decrease. The chapters on the pathological manifestations of abnormal carbohydrate and fat metabolism, on infection and on the pathology of coma have a similar significance for the clinician; as Dr. E. P. Joslin writes in his foreword: "This is a pathology of and for the living almost as much as of the dead."

Some of the old problems remain problems still. There is little that is fresh on the nature and causation of diabetic neuritis. The insulin-resistant case remains a mystery. Fat atrophy at the site of injection is not yet clearly understood. Diabetic retinitis is not even mentioned. For the patient who, dying of diabetic coma, presents no lesions referable to diabetes—the thorn in the flesh of all diabetic pathologists—Dr. Warren has an ingenious explanation concerning variations in pancreatic blood supply, which is as yet little more than a theory; further development of this theory will be watched for with the keenest interest.

An admirable summary of technical methods of value to the pathologist dealing with diabetic material concludes a presentation of work painstakingly carried out and lucidly set forth. All who have any part in the handling of diabetes cannot but be grateful for the book.

CLINICAL PATHOLOGY.

"OF making many books there is no end and much study is a weariness of the flesh." The lament of "The Preacher" in the book of "Ecclesiastes" cannot be said to be applicable to books dealing with clinical pathology, for in this rapidly evolving specialty there is by no means a surfeit of good books. In Todd and Sanford's "Clinical Diagnosis by Laboratory Methods," the seventh edition of which has now appeared, will be found an excellent text book for medical students and a valuable *vade mecum* for the specialist in clinical pathology.[1]

As explained in the foreword, the book has grown with the development of the subject from a set of lecture notes prepared for his students by Professor James C. Todd, of the School of Medicine, University of Colorado. Rendered unable by impaired health to cope with repeated revisions alone, Professor Todd has sought and obtained the collaboration of Dr. Arthur H. Sanford, Professor of Clinical Pathology, University of Minnesota (The Mayo Foundation) and Head of the Section on Clinical Laboratories, Mayo Clinic.

In the section of the book devoted to bacteriology the descriptions of the various microorganisms are necessarily brief. Bacteriological methods are well described and judiciously selected, and the details of technique in serodiagnosis are well worth the perusal of laboratory workers in serology. On pages 686-687 are tables giving the new names for bacteria proposed by the Society of American Bacteriologists, but the nomenclature in the book suggests a disinclination on the part of the authors to abandon many of the old terms. Thus, while they describe the spirochæte associated with the fusiform bacillus in Vincent's infection as *"Borrelia vincentii,"* they do not present the typhoid bacillus as the *"Eberthella typhi"* or introduce the meningococcus as *"Neisseria intracellularis."*

In the discussion of the leptospira, the *Leptospira icteroides*, of Noguchi, is included with the statement that it is believed to be the causal agent in yellow fever. It was perhaps a little surprising to find no reference to the significant criticisms which have been directed against the *Leptospira icteroides* in this connexion, the force of which was recognized by the distinguished Japanese scientist himself.

Hæmatology is particularly well treated. In the chapter dealing with the isohæmagglutination groups the authors announce their adherence to the Moss system of numbering, and in general give an excellent discussion of isohæmagglutination in its relation to blood transfusion.

In the chemistry of the blood the methods of Folin and Wu are given pride of place, but it seems anomalous that in the procedures detailed for the estimation of the blood sugar the widely used method of McLean finds no place. Methods requiring two cubic centimetres of blood for each determination are given precedence and apparently resort has to be taken to micro-methods, with admitted loss of accuracy, when such a quantity of blood is not readily obtainable. In dealing with children and adults with small veins, the method of McLean, which requires 0·2 cubic centimetre of blood and necessitates a needle prick only, has obvious advantages which in our opinion should have secured it a place in the book.

The book is admirably printed, with 347 illustrations, 29 of which are in colour, distributed through 765 pages. The ultra-American spelling of certain words at first took us unawares, but eventually we recognized a familiar implement for the measurement and transference of liquids disguised as a "pipet."

It can well be understood that any attempt to have included the subject of pathological histology in the book might have resulted in a much larger volume than the authors desired, but no one without a knowledge of pathological histology can properly describe himself as a clinical pathologist. In our opinion the present volume, "Clinical Diagnosis by Laboratory Methods," supplemented by one of equal merit in pathological histology would supply all the needs of the specialist in clinical pathology.

[1] "Clinical Diagnosis by Laboratory Methods: A Working Manual of Clinical Pathology," by J. C. Todd, Ph.B., M.D., and A. H. Sanford, A.M., M.D.; Seventh Edition; 1931. Philadelphia: W. B. Saunders Company; Melbourne: James Little. Royal 8vo., pp. 765, with 347 illustrations, 29 in colours. Price: 36s. net

VENEREAL DISEASE.

THE second edition of David Lees's "Practical Methods in the Diagnosis and Treatment of Venereal Diseases" follows its predecessor after an interval of four years.[1]

This excellent volume has been written for the student and practitioner and embodies the methods of treatment practised with success by the author at the Edinburgh Royal Infirmary and elsewhere.

To keep the new edition abreast of modern progress, recent advances which have proved themselves of value, have been added without appreciably increasing the size of the volume. The book is in two parts, the first dealing with syphilis and the second with gonorrhœa. The field is fully covered, and sound principles of diagnosis and practical details of modern treatment will be found throughout. A new chapter on cardiovascular syphilis is included, and the chapters on neurosyphilis and its treatment have been largely rewritten and brought abreast of modern teaching by including a statement of the effect of treatment by "Tryparsamide," malaria and other forms of pyrotherapy.

The standard of cure of syphilis and gonorrhœa is fully discussed and the fact is repeatedly stressed that the disappearance of the signs and symptoms of these diseases does not mean the cure of either.

The illustrations and general arrangement are excellent, and as the author fully knows his subject, this volume may be confidently recommended to those who desire information about the important diseases with which it deals.

[1] "Practical Methods in the Diagnosis and Treatment of Venereal Diseases for Medical Practitioners and Students," by David Lees, D.S.O., M.A., M.B., D.P.H., F.R.C.S., M.R.C.P., with introduction by W. Robertson; Second Edition; 1931. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 654, with illustrations. Price: 5s. net.

The Medical Journal of Australia

SATURDAY, OCTOBER 17, 1931.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

RADIUM AND CANCER.

During recent months malignant disease has been referred to on several occasions in these columns, more particularly in reference to the Second Cancer Conference held at Canberra last March. Since then some of the contributions to that conference which had a clinical bearing, have been published in this journal. These papers show that serious attempts have been made in many centres in the Commonwealth to use radium and to demonstrate its usefulness in the treatment of malignant disease. There is a considerable difference between the complete determination of the efficacy of radium and the demonstration of what it can do in certain circumstances. It must be admitted that the limitations to the use of radium have not been accurately determined. Medical investigators know that certain types of growth are susceptible to radium rays and that others are not so readily influenced. Workers in several countries in Europe have reported the most astounding results in properly controlled and large series of cases. These results must be accepted. It is not necessary, as has been suggested by certain conservative persons of purblind insularity, that the spade work should be repeated in every country before it is regarded with favour by workers in that country. Were this necessary, it would be a long time before radium treatment could hope to be standardized in Australia. There can be no doubt that in due course the available information will be sufficient to justify the laying down of standards in treatment. The papers read at the Cancer Conference will give medical practitioners in Australia some idea of what may be expected as the immediate result of radium treatment; and this alone, if no other reason were forthcoming, would be an adequate reply to those who would question the wisdom of publishing results at such an early stage.

There are many other points that might be discussed in connexion with the proceedings of the last Cancer Conference. Two only will be mentioned. The first has to do with diagnosis and the choice of treatment. Medical practitioners have no need to be reminded of the importance of early diagnosis. They know that early treatment, whether that treatment be by radium, X rays or surgical removal, alone offers the patient hope of cure. They may not know how they may obtain help from the organization at present existing in Australia for the control of cancer. Cancer treatment centres or clinics have been established at the principal hospitals in the main centres of population. Radium purchased by the Commonwealth in 1928 has been distributed on loan to these centres, and in many instances they have been provided with X ray plants for purposes of treatment. A consultative committee, composed of surgeons, radiologists, pathologists and other specialists, meets regularly at these clinics. Patients sent to the clinic are seen by the members of this committee, the patient's condition is discussed and advice is given in regard to the form of treatment likely to be successful. Treatment, whatever its nature, is immediately available at the clinic. Medical practitioners are therefore urged to make themselves acquainted with the working of these clinics and to use them. They will receive help in diagnosis and advice as to treatment. Further, when special measures are called for, they will be able to put the patient into the hands of experts; this they owe to the patient.

The Commonwealth Department of Health acts as collator of records for the cancer treatment centres. The forms used are uniform (some were

exemplified in recent issues of this journal) and are in accordance with the requirements of the League of Nations Health Organization. Medical practitioners can help in no small way by giving full information on the death certificates of persons dying of cancer. Analysis of death certificates shows that indefinite and often misleading terms are used, and that the anatomical site of the disease is often not indicated. General terms, such as cancer, malignant disease and malignant tumour, convey little information and apply equally to such widely dissimilar conditions as carcinoma and sarcoma. The use of an indefinite term on a death certificate would indicate that the attending practitioner had not gone to the trouble of making a proper diagnosis. He should above all avoid the stigma of carelessness.

Current Comment.

RADIOLOGY OF THE HEART.

Radiological examination of the heart is fairly extensively practised in America and Europe; in the United Kingdom and in Australia little interest appears to be taken in it. This is remarkable, in view of the great progress that has been made in radiology within recent years. Perhaps the clinician is jealously endeavouring to retain sole rights to the one remaining field in which he feels he can discover more by medium of his senses than can the radiologist with his chemicals and machinery. But the clinician's findings are often not nearly so accurate as he believes. Mapping out the precordial area by means of percussion requires skill and experience; there are so many possible sources of fallacy that it is doubtful whether it ever can be done with accuracy. The clinician may fail to recognize even gross enlargement of the heart under certain conditions, and under other conditions he may diagnose hypertrophy when the size of the heart is normal. Of course, orthodiagraphy is a procedure, the value of which, in the diagnosis of aortic aneurysm, is generally recognized. This method of radiological examination of the heart is also usefully employed in the diagnosis of cardiac hypertrophy. In a paper that he read before the Section of Electro-therapeutics of the Royal Society of Medicine, G. Grant Allan makes a plea for the more general use of cardiac radiology and discusses various methods.[1] He suggests that the chief reason why physicians and radiologists have been indifferent to radiological examination of the heart, is that there has been no standardized method of examination. Among other methods employed in the estimation of cardiac hypertrophy he mentions the determination of what is known as the "cardio-radiographic index." The patient is examined by means of a fluorescent screen; a narrow vertical beam of X rays is allowed to pass antero-posteriorly through the body, tangentially to the left border of the heart; the position of the left border is marked on the screen. The patient and the screen are kept in the same positions, and the source of the beam is shifted to the right for a distance of ten centimetres; again the beam is directed past the left border of the heart. The edge of the shadow now thrown on to the screen is to the left of the original mark. The distance between the marks is the cardio-radiographic index. The figure, of course, is higher when the left ventricle bulges posteriorly. It is claimed for this method that it reveals early hypertrophy of the left ventricle. No doubt it is of some value; but there are obvious sources of error, and it is questionable whether any reliance should be placed on it unless the figure is considerably above normal.

When the X ray tube is close to the body, screen examination and radiography are apt to give fallacious results on account of the distortion of shadows due to diverging rays. To overcome this difficulty Köhler introduced teleradiography: that is, radiography with the patient two metres or more from the tube. At this distance the effect of the divergence of the rays is very silght, and, if greater accuracy is necessary, the aberration may be allowed for. The disadvantage of this method is that specially powerful apparatus is necessary.

Allan carefully examines the heart, aorta and mediastinum, in antero-posterior and oblique views, by means of the fluorescent screen; he then makes an estimation of the cardio-radiographic index; finally, he makes a teleradiogram, placing the patient at a distance of two metres from the tube. A tracing of the radiogram is made and a vertical line is drawn through this in a position corresponding to the mid-sagittal plane of the spine. The sum of the lengths of perpendiculars dropped on to this line from the most distant points on the left and right borders respectively, equals the transverse diameter of the heart. A line joining points corresponding to the upper limit of the right auricle and the apex represents the longitudinal diameter. Perpendiculars are drawn to this line from a point corresponding to the junction of the right border of the heart and the diaphragm, and from a point on the left border representing the upper limit of the left ventricle. The sum of the lengths of these perpendiculars is taken as the diameter of the base of the heart. The area of the heart shadow is then measured by means of a planimeter. Allan has found, however, that when the heart is hypertrophied, some of the landmarks are difficult to distinguish; he therefore measures the whole shadow of the heart and aorta (that is, the projection of heart and aorta). He compares this area with the total surface area of the patient's body. He finds that normally the relation varies only

[1] *Proceedings of the Royal Society of Medicine*, July, 1931.

slightly with the individual and remains approximately as 0·7 to 100. When there is gross hypertrophy the figure may be as high as 1·94 *per centum*. He admits that he has not studied a sufficient number of normal or abnormal subjects to enable him to lay down definite standards.

By means of this method of examination, information is also gained concerning the direction of the cardiac axis. This is valuable, and should be correlated with the electrocardiographical findings.

Allan's contribution is of considerable importance and more than ordinary interest; but the figures he obtains by comparing the area of the normal person's heart shadow (incidentally he calls it the area of the heart) with the area of the body surface, should not be accepted until they can be confirmed by further investigation. Examination should be made of tall, short, thin and stout persons whose hearts are normal. It would be surprising if the figure remained constant. As was pointed out in the discussion following the reading of the paper, it would perhaps be wiser to consider heart and aorta separately; for disease may be present in the one and not the other.

If Allan has done nothing else, he has shown that radiography is likely to become an important aid in the diagnosis of cardiac disease.

THE TRANSPLANTATION OF URETERS.

The transplantation of the ureters into the intestine is an operation which was undertaken originally for exostrophy of the bladder. It may be undertaken in the presence of inoperable cancer of the bladder, prostate or urethra, or as part of the surgical treatment of operable cancer of these organs. Involvement of neighbouring organs, such as the uterus, by extensive malignant disease may call for transplantation of the ureters and other possible indications are tuberculosis of the urogenital system and trauma. In the early days the immediate post-operative mortality was high, but later developments and improvement have reduced the mortality considerably. The name of Coffey is preeminent in this branch of surgery. His technique has been followed by surgeons the world over and successful operations, often in the face of great difficulty, have been reported in this journal by more than one Australian surgeon.

In Coffey's original operation an incision was made through the peritoneal and muscular coats of the intestine and through the submucous layer. Sutures were passed through the peritoneal and muscular coats on each side of the incision. Sutures inserted into the cut end of the ureter were passed, together with the end of the ureter, underneath the sutures in the bowel wall. A stab wound was made in the mucosa of the bowel and the traction sutures on the end of the ureter were passed into the lumen of the bowel and out through the intestinal wall. The ureter was thus anchored to the bowel wall. The sutures in the wall of the bowel were then tied. The ureter was "tacked" to the peritoneum of the intestine near its point of entrance. Coffey subsequently described two modifications of his original procedure. In his second plan he introduced cleansing of the bowel and the use of ureteral catheters. After the bowel is irrigated, first with water and then with a solution of mercurochrome, the bowel is packed with gauze, inserted through a sigmoidoscope. An incision is made in the bowel wall (peritoneal coat and muscular layer). Finally, the mucosa is incised as in the first type of operation, and the ends of catheters inserted into the ureters, are sutured to the gauze. By withdrawal of the gauze through the anus the catheters and ends of the ureters are drawn into the bowel. The wall of the bowel is then sutured. In Coffey's third plan the ureter is transfixed with a needle carrying a strong thread, the needle picks up the mucosa and the suture is tied tightly so as to strangle all the tissues within its grasp.

A valuable piece of experimental work has recently been carried out at the Department of Urology of the Royal Prince Alfred Hospital, Sydney, by P. N. Walker-Taylor.[1] Walker-Taylor has tried to overcome the risk of infection of the peritoneum by the fact that in operations of the Coffey type an incision is made into the lumen of the bowel or a needle is passed from the peritoneal cavity into the lumen of the bowel and back again into the peritoneal cavity. He has implanted 106 ureters into the bowel of 77 dogs. The fundamental principle of his work is the adoption of the "closed tunnel" technique. In this method a longitudinal tunnel is made in the submucous layer of the bowel. Walker-Taylor states that he found that with the Coffey technique infected material found its way from the lumen of the bowel to the neighbourhood of the stitches and caused peritonitis. Walker-Taylor describes the "open tunnel" technique and the "closed tunnel" technique. In the open tunnel an incision is made at the distal end of the tunnel in the submucous layer of the bowel; in the closed tunnel no distal incision is made. Walker-Taylor then describes his "aseptic irreversible tunnel" technique. With this method a metal cylinder with an obturator is introduced into the anus. With a piercing instrument or a mucosal punch an opening is made into the lumen of the bowel and the perforating instrument, to which the end of the ureter is attached, is drawn out of the anus through the metal cylinder. It is obvious that in this way no instrument is inserted into the bowel from the peritoneal cavity and drawn back into it again; there should be no soiling of the peritoneum.

On paper Walker-Taylor's operation appears to be ideal. It remains to be seen how it will serve in surgical practice. Surgeons are advised to hasten slowly. Operations suited to the abdomen of the dog are not always suitable for a human being. At the same time Walker-Taylor's technique must be studied and carefully followed before his method is condemned.

[1] *The Australian and New Zealand Journal of Surgery*, September, 1931.

Abstracts from Current Medical Literature.

MEDICINE.

Insulin Angina.

A. E. PARSONNET AND A. S. HYMAN (*Annals of Internal Medicine*, April, 1931) discuss the proper management of that group of diabetic patients suffering from such cardiovascular complications as myocardosis, *angina pectoris* and coronary arterial disease of greater or less degrees of severity. The clinical picture of the middle-aged individual with a long previous history of diabetes, held more or less in check by a moderate though not absolutely scientific dietary *régime*, who is suddenly projected from a state of hyperglycæmic complacency into the throes of an acute coronary attack following an injection of insulin, is a not unfamiliar incident. The authors state that Banting himself, in a conversation with one of them, stressed the fact that diabetes today is a much over-treated disease. The incidence of diabetes in coronary thrombosis is relatively high. In the authors' series of 89 cases of coronary thrombosis there were 22 patients with definite diabetic symptoms and of this group seven had severe coronary seizures following the initial first day dosage of insulin. The clinical records and electrocardiograms of four of these cases, one of which had a fatal termination, are given by the authors. The mechanism of the seizures is discussed briefly. An interference with the glycogen metabolism of the heart is postulated. The authors conclude by emphasizing the necessity for a complete examination of the heart, including electrocardiographic studies in cases of diabetes associated with arteriosclerosis before the institution of insulin therapy. Further, when diabetes is complicated by known cardiovascular disease, they studiously avoid the administration of insulin until dietary measures have failed to produce results commensurate with safety. Finally, in none of the cases quoted did the blood sugar levels fall below the limit of safety.

Disturbances of the Parathyreoid Gland.

J. C. MEAKINS (*The Canadian Medical Association Journal*, May, 1931) discusses disturbances of the parathyreoid gland from the clinician's viewpoint. The parathyreoid gland is intimately concerned with the function of calcium regulation. Parathyreoid tetany is a condition which occurs subsequent to removal of the parathyreoid glands or to their injury or degeneration from some other abnormal cause, but tetany is a syndrome which may occur in any condition in which there is a low blood calcium or even a normal blood calcium with a prolonged negative balance. Such conditions are to be found in steatorrhœa with megacolon, infantile rickets and osteomalacia. The relationship between parathyreoid tumours and diseases of bone is discussed with reference to *osteitis fibrosa* (von Recklinghausen's disease). This condition occurs more commonly in women than in men, and generally between the ages of thirty and fifty-five. The patients complain of pains in the bones. Spontaneous fractures are common. There is an increase of the blood serum calcium varying from 13 to 23 milligrammes per 100 cubic centimetres, and a reduction of plasma phosphorus to between 1·0 and 2·7 milligrammes, and in all cases an increase in the calcium output. It would appear as if this condition were the opposite of that found in parathyreoid tetany, except that there is the additional factor of pronounced bone destruction. The resemblance between osteomalacia and *osteitis fibrosa* is only superficial, as tetany occurs in osteomalacia. In nephrosis there is a definite lowering of the blood calcium, and some patients suffering from this disease respond favourably to the use of parathyreoid extract, not only in so far as the restitution of the blood calcium is concerned, but also as regards the disappearance of the anasarca. There is no evidence, however, that the disease is primarily due to a disturbance of the function of the parathyreoid gland.

Intrapericardial Aneurysms.

T. MARTINI AND M. JOSELEVICH (*Revista Médica Latino-Americana*, December, 1930) discuss the diagnosis of intrapericardial aneurysms of the aorta. Inspection may reveal pulsation which is not expansile in the third and fourth intercostal spaces on the right side of the sternum or in the second or third left intercostal spaces close to the sternum; palpation often gives a systolic and sometimes a diastolic thrill; percussion shows dulness to the right and above the normal cardiac dulness. A to-and-fro murmur is heard in the third or fourth interspaces to right or left of the sternum; it is propagated in every direction. Compression of the *vena cava* and right auricle may cause congestion of the face and of the liver respectively, possibly œdema of the legs. Compression of the pulmonary artery may give rise to dilatation of the right ventricle; the heart may be displaced to the left. The incompetent aortic valve causes hypertrophy of the left ventricle. The anuerysm may rupture into an adjacent artery or vein or into one of the chambers of the heart with dramatic symptoms of suffocation, pain and orthopnœa. The usual symptoms of these aneurysms are similar to those occurring in extrapericardial dilatations, and the diagnosis is made mainly on the clinical signs.

Paroxysmal Hypertension.

M. VILLARET (*La Presse Médicale*, March 18, 1931) discusses paroxysmal arterial hypertension. This condition may be due to one of four causes: adenoma of the medulla of the suprarenal gland, injection of adrenalin, toxins within the body, such as alcohol, nicotine and lobeline, and nervous excitation of adrenalin secretion. It is well known that stimulation of the central end of the pneumogastric nerve will cause a paroxysm of hypertension, and hypertension of this type has been noted in patients with tumours affecting this nerve. Stimulation of sensory nerves may have a similar effect as in renal colic, stretching the sciatic nerve and during dental treatment; during dental treatment dangerous symptoms may arise due to hypertension; they may be relieved by venesection. Cerebral compression and cerebral embolism may be associated with hypertension due to stimulation of the adrenalin secretion, and asphyxia from any cause may have a similar effect and may give rise to hæmorrhages or thrombosis. Muscular contraction may cause temporary hypertension and emotional disturbances have a similar effect. All these forms of paroxysmal hypertension are due to hyperadrenalinæmia. Apart from these, ephedrine, ergotamine, pituitrin (or oxytocin), tyramine and some mineral waters may cause hypertension; and in Graves's disease and some cases of acute and chronic nephritis transient hypertension may ensue. In plumbism and eclampsia similar effects occur, and *angina pectoris* is often associated with paroxysmal hypertension. In acute œdema of the lungs the pressure is temporarily raised, and during the gastric crises of tabes the same phenomenon has been observed. In chronic hypertension also a great increase of blood pressure of short duration may occur. Treatment should be directed to the removal of the cause when possible; sedatives, such as bromides, barbitone or morphine, may be indicated. Rest is essential; amyl nitrite, trinitrin and acetylcholine may be used. Venesection is indicated in eclampsia and acute œdema of the lungs.

Arteriosclerosis.

R. D. EVANS (*California and Western Medicine*, March, 1931) discusses the pathology and ætiology of arteriosclerosis; arterial changes due to syphilis and arteriosclerosis are not included. Five conditions are recognized, senile ectasia, atherosclerosis affecting the aorta and larger arteries mainly; hyalinization, arteriocapillary fibrosis, medial hypertrophy and *endarteritis obliterans* affecting the smaller vessels. Senile ectasia affects the aorta mainly, elastic tissue deteriorates and connective tissue increases so that the aorta becomes overstretched, tortuous and dilated. Atherosclerosis is associated with deposition of fat in the intima; such a change is found in 35% of people at twenty years and in 100% over forty years of age. Cholesterin esters are deposited and later fat droplets accumulate in the intima and a raised patch is formed. In later years grey or yellow elevated plaques occur in the aorta, lime salts are deposited and

ulcers may appear from loss of intimal covering. The cholesterin which is deposited, splits off fatty acids and calcium soaps are formed. Mönckeberg's sclerosis is merely an expression of arteriosclerosis in its wider sense; it affects mainly the limbs, calcification of the media occurs, giving a deposit comparable in form to the rings of the tracheal cartilages. In the small arteries of the spleen, brain, kidneys and uterus hyalinization occurs, a homogeneous refractile mass is deposited in the intima and media. Amyloid changes fall in this group. The arteriocapillary fibrosis of Gull and Sutton occurs in the arterioles of the spleen, kidneys, liver and other organs, probably in all the arterioles and small vessels; hyaline and fatty changes occur and connective tissue proliferation narrows the vessels. *Endarteritis obliterans* is due to proliferation of the connective tissue of the intima; it occurs as an involutionary change normally in the ovary and elsewhere. Infectious diseases, increased blood pressure of short or long duration, and degeneration due to the age of the individual are advanced as causes of arteriosclerosis. Increased cholesterin content of the plasma may be a factor. Arteriocapillary fibrosis is always associated with raised blood pressure, it involves the smaller arteries and capillaries and occurs at a younger age than senile arteriosclerosis. High protein and high cholesterin-containing diets have been shown to produce atherosclerosis in animals, and high protein diets of the acid ash type have a similar effect. A diet deficient in eggs, milk, fresh cooked vegetables, fruit and lettuce is said to be a frequent precursor of degenerative diseases; these foods contain vitamins, residue and alkaline minerals. More concentrated foods, high in carbohydrates and acid minerals, compose the main diet of the subjects of degenerative diseases. The endocrine glands may play a rôle in causation of arteriosclerosis, sympathetic stimulation with increased output of adrenalin may increase the blood cholesterol. In diabetes lipoid metabolism is often disturbed and may be related to the arteriosclerosis of the large vessels and of the Mönckeberg type in this disease. Thyreoidectomy, splenectomy and gonadectomy hasten the deposition of cholesterol in the intima in rabbits on a high cholesterol diet. Castration slows the excretion of cholesterol. Typhoid fever and diphtheria cause medial injury. Heredity is a potent predisposing factor. Alcohol and tobacco are not proved causative agents. Lead, on the contrary, causes vasoconstriction and both intimal and medial injury.

Whooping Cough.

E. Stettner (*Deutsche Medizinische Wochenschrift*, January 30, 1931) reports on the treatment of whooping cough by specific vaccines. He claims vastly better results than have hitherto been obtained by this method of treatment; these he attributes chiefly to the size of the doses, which should be 2,000, 4,000, 6,000 and 8,000 million organisms (*Bacillus pertussis*) injected intramuscularly. In almost every case a definite febrile allergic reaction was observed; in early cases this was commonly observed after the third injection, but if the condition was more advanced, the reaction occurred proportionately earlier, that is, after the second or even the first injection. Results were more satisfactory in the earlier cases, but even in protracted cases marked improvement followed the course of injections.

Carbohydrate Tolerance in Febrile Conditions.

Sick (*Münchener Medizinische Wochenschrift*, April 10, 1931) has investigated carbohydrate tolerance in a series of febrile patients in all of whom *diabetes mellitus* was excluded by subsequent control investigations. Using the Staub technique, which differs from that of MacLean in that two doses each of thirty grammes of glucose are administered to the fasting patient with an interval of one hour between the doses, the author finds that the resultant blood sugar curve closely resembles that found in mild and early diabetes. There is, however, in the febrile cases a concurrent rise in renal threshold, so that in only one case out of twelve in which the blood sugar rose over 180 milligrammes *per centum* did glycosuria appear. That the abnormality of the curve is due to the infection and not to the fever is suggested by the persistence of the high values in certain cases for some days after the temperature has subsided, and also by the absence of these findings in the hyperpyrexia of exophthalmic goître. Hepatic disturbance is suggested as providing an explanation for the phenomenon.

Intravenous Use of Extract of Liver.

W. B. Castle and F. H. L. Taylor (*The Journal of the American Medical Association*, April 11, 1931), in a preliminary communication, give the results of observations on the intravenous injection of liver extract. The original "fraction G" of Cohn was used on account of its relative ease of preparation and because the blood pressure reducing substance was found difficult and expensive to eliminate. The depressor effect was found to persist for only one or two minutes and to be easily controlled by varying the rate of injection. Observations were first made on cats and rabbits. Apart from transient effect on blood pressure, 0·2 gramme of "fraction G" per kilogram of body weight in aqueous solution had no deleterious action on rabbits. For injection in patients suffering from pernicious anæmia an extract containing a little over 0·1 gramme per kilogram of body weight of "fraction G" was dissolved, after washing with ether, in physiological solution of sodium chloride. In patients injected with this material both systolic and diastolic pressures were decidedly lowered if the extract was injected faster than at the rate of two cubic centimetres per minute. The more anæmic the patient, or the lower the original blood pressure, the more marked was the effect, but in all instances the pressure returned to its original level within one or two minutes. The results of a single injection in each of two patients of the amount of material derived from 100 grammes of liver were maximal reticulocyte responses of 27·8% and 25·4% and a gain within ten days of a million red cells from initial levels of 0·98 and 1·49 million red blood cells per cubic millimetre respectively. One of these patients had previously received liver extract derived from 300 grammes of liver daily for ten days with no significant change in the red blood count in twenty days. In a third patient the extract derived from fifty grammes of liver produced on the sixth day 8·4% of reticulocytes and a gain of 0·5 million red cells from an initial level of 1·29 million within ten days. The possible ætiological and therapeutic significance of the observations is suggested.

Schottmüller (*Münchener Medizinische Wochenschrift*, March 13, 1931) describes his experience in pernicious anæmia of the use of "Hepatopson," a liquid extract of liver. He found that remarkable improvement occurred in a few days after injection of this preparation. The author draws attention to the smallness of the dose required as compared with that necessary for oral administration. One to three cubic centimetres of the preparation, corresponding to ten to thirty grammes of liver substance, given every day, were found sufficient. By mouth an amount of extract corresponding to 500 to 2,000 grammes of fluid liver are necessary to produce the same result. The author advocates the injection treatment for patients with a very low blood count and anorexia, and for patients in whose treatment a blood transfusion is considered necessary as a life-saving measure. For the milder forms of pernicious anæmia the oral administration is advised as being sufficient.

Diagnosis of Pancreatitis.

P. Kaczander (*Deutsche Medizinische Wochenschrift*, June 26, 1931) reviews the methods of diagnosis of pancreatic lesions. He emphasizes the value of the Wohlgemuth diastase urinary test in arriving at an early diagnosis. This was tried in 305 various abdominal conditions, and a high count was noted with thirty-five. The upper limit of a normal result to the test was taken as 64 units. Of the patients with a high count 13 were suffering from conditions pancreatic in origin; four conditions were acute and nine chronic. The remainder were caused by gall bladder lesions and appendicitis. Despite these exceptions the author believes that the diastase test is of great value in the early diagnosis of these acute abdominal crises.

Special Articles on Aids to Diagnosis.

(Contributed by Request.)

IV.

THE DIAGNOSTIC USE OF TUBERCULIN.

The tuberculin test depends on the observation which Robert Koch made in guinea-pigs and confirmed in men, that an animal infected with tuberculosis reacts to products of the tubercle bacillus differently from a non-infected animal.

Many methods of employing the test have been devised, as by Von Pirquet, Calmette, Wolff-Eisner, Mantoux, but they are all examples of Koch's phenomenon. This article describes only the original and most informative one, the subcutaneous. It is used in cases of chronic pulmonary disease in which no tubercle bacilli can be found in the sputum, and in all cases with vague symptoms in which tuberculosis is a possibility. "Many slight, ill-defined ailments are due to a local, unrecognized tuberculosis of the lung."

It should not be used when there is any fever, and particularly not in meningitis.

Single doses of tuberculin may be purchased dispensed ready for use, but it is wiser for the physician to measure his own afresh.

The material employed is Koch's old tuberculin or Koch's albumose-free tuberculin, as manufactured and standardized by Meister Lucius and Brüning, of Hoechst on the Main; it is put up in one and in five cubic centimetre bottles. Old tuberculin is made also by Burroughs Wellcome and Company, and by the Commonwealth Serum Laboratories. It is prepared from cultures of living tubercle bacilli, but of these it contains none, for it is filtered through a germ-proof filter, concentrated to one-tenth of its bulk by boiling and diluted to a standard strength with glycerine.

Before the bottles are opened the neck and stopper should be passed with a rotary movement for about two seconds through the flame of a spirit lamp or of a Bunsen burner.

Individual doses are suitably measured with a glass pipette about three millimetres in diameter, calibrated in one-hundredths of a cubic centimetre, so that 0·1 cubic centimetre fills about 70 millimetres. This may be sterilized by washing it out with nearly boiling water and then drying it in the flame of the spirit lamp. Syringes may be obtained graduated in one-hundredths of a cubic centimetre and holding one cubic centimetre in all; some find these more convenient than the pipette for measuring doses.

Doses and dilutions may be temporarily stored in small glass bottles holding about two cubic centimetres and fitted with rubber stoppers.

The syringes, bottles and stoppers are sterilized by boiling.

A convenient rack for resting pipettes and syringes may be made by folding longitudinally a very stiff piece of note paper about twelve centimetres long and four centimetres wide; this is then nicked in two or three places along the fold and allowed to stand on its partly opened edges.

The diluent employed is either 20% glycerine in water or 0·5% carbolic acid in normal salt solution, that is, 44 grains of carbolic acid and 75 grains of sodium chloride in one pint of distilled water. This should be boiled and dispensed in a boiled glass bottle with a boiled glass stopper.

In making the dilutions the mouth and stopper of the bottle of diluent are flamed in the spirit lamp, the stopper is removed and five or six cubic centimetres are poured into a sterilized chemical test tube and boiled over the lamp; the test tube is then stoppered with a plug of dry sterilized cotton. Now flame the mouth and stopper of a bottle of undilute tuberculin, remove the stopper and with a sterilized pipette or syringe draw off 0·1 cubic centimetre and put this in a sterilized test tube; with similar precautions add to it 0·9 cubic centimetre of the sterilized diluent and boil over the flame of the spirit lamp; decant it off with a clean sterilized pipette or syringe into a sterilized bottle; flame the mouth of the bottle, flame the stopper and stop the bottle. From this first dilution (one in ten) a second dilution (one in a hundred) may be made and from this a third (one in a thousand) and so on.

Make sure that the temperature of the patient to be tested is normal, that is, no higher than 37·2° C. (99° F.) for a day or two. If there is no nurse available to take the temperature, it will be necessary to take it yourself or teach the patient or some responsible house mate how to do it. Convenient times for taking the temperature are 7 and 11 a.m. and 2, 5 and 8 p.m. The thermometer should be kept under the patient's tongue with the lips closed for five or six minutes in the summer and for six or eight minutes in the winter.

The full test dose for an adult is 0·01 cubic centimetre, but this must not be given till it is known that the patient does not react to smaller doses. We therefore begin with 0·001 cubic centimetre (sometimes called one milligramme) or a smaller dose (approximately proportional to weight) for a child, as 0·0001 for a baby of two years, 0·0003 for a child of six, 0·0005 for a child of ten. The dose is measured with aseptic precautions as described above and placed in a sterilized syringe.

One requires some sterilized absorbent cotton, plain, not medicated, some absolute alcohol (or rectified spirits of wine) and adhesive rubber plaster cut into pieces two or three centimetres by one or one and a half centimetres.

The site of election for the injection is the outer side or back of the forearm, two to four inches below the elbow, or of the upper arm, some two inches or more above the elbow. Choose a spot free of large veins, vaccination scars or other obstructions, rub it briskly with a pledget of cotton soaked in the alcohol and make the injection into the subcutaneous tissue; flame a small dry pledget of cotton, shake it briskly to put it out, and press it thus freshly sterilized on the puncture immediately after withdrawing the needle. Do not massage the injected area, but replace the cotton pledget by a piece of the rubber plaster the face of which has just been sterilized in the flame. No further dressing is required.

The patient should keep quiet and take the temperature as before, and in addition at any other time he may feel feverish. It is not necessary for him to remain in bed, but he should be near home, so that he may go to bed in case he reacts severely.

He should be seen two days later so that the result of the test may be observed. The reaction usually begins in about six or twelve hours, or if delayed, in twenty-four or thirty-six hours. If it occur, it is always within forty-eight hours. If an injection be given intravenously, instead of subcutaneously, there will be no local reaction, but there will be, in a sensitive patient, an early and severe general reaction. The reaction may appear in three forms:

1. The local reaction is seen at the site of the injection as a hard, red, hot, painful swelling. It may be some two, four or six centimetres in diameter. Very rarely larger reactions occur even ten or twelve centimetres in diameter, intensely red, with a surrounding less intense œdema so as to suggest suppuration, but this never occurs. No dressing is needed, all will subside in a few days, but some patients are comforted by boracic or other foments.

2. The general reaction consists in fever, with the usual accompaniments, headache, malaise, pains in the limbs and joints resembling an attack of influenza. The temperature may rise to 37·8° C. (100° F.) or 38·3° C. (101° F.) or even more. It rarely continues more than six or twelve hours.

3. The focal reaction occurs at the site of the disease and appears as an increase in the symptoms and signs. Thus in tuberculosis of the lung there may be pleural pain, cough and expectoration; in tuberculosis of the knee or cervical glands, pain and swelling; in tuberculosis of the kidney, pain in the loin, frequency of micturition and pyuria.

It is very unusual for the reaction to the initial dose to be more than slight.

If there is a definite reaction, there is no occasion to test further.

If there be no reaction, a second dose is injected two days after the first. The ideal amount of this is five times the first, thus 0·005 cubic centimetre for an adult.

If there be no reaction to the second dose, a third is given two days later again. This will be twice the second, thus 0·01 cubic centimetre (sometimes called ten milligrammes).

If a doubtful reaction occur as a trifling soreness at the site of the injection, a vague increase of symptoms or signs, or a rise in temperature to no more than 37·5° C. (99·5° F.) the previous dose should be repeated after an interval of two days. Thus, if there have been no reaction to 0·001 cubic centimetre and a doubtful reaction to 0·005 cubic centimetre given two days later, the second dose should be repeated after two days, and if no reaction to it, the third given after another two days. But it often happens that whereas only a doubtful reaction occurred after a dose, a very definite reaction occurs on repeating the same dose two days later; this increase of sensitiveness on repeating the dose was considered by Koch to be the most typical reaction, and clinical experience confirms him.

Interpretation of Results.

In interpreting the results of the test there are several points for consideration.

1. When no reaction has occurred to the full dose, then either the patient has never been infected with tuberculosis or has been previously immunized by repeated injections of tuberculin so as no longer to react. A third possibility is that the patient is suffering from severe and rapidly advancing tuberculosis so that the dose injected makes little difference to the large amount of tuberculin he is already absorbing from his own lesion; but, of course, the appearance of such a patient and the fever which he will probably exhibit should have protected him from the application of the test.

2. If a reaction occurs, the patient has undoubtedly been infected with tuberculosis; but this does not mean that he cannot possibly be suffering from some other disease; although he has tuberculosis, it may not be his most important illness.

Sometimes a local reaction occurs alone. This shows that the patient has been infected with tuberculosis, but it does not show that he now suffers from it; his disease may be quiescent or healed.

Sometimes a local and a general reaction occur without a focal. This shows that the patient has been infected and that the disease is still active. It does not show where in his body the disease is.

It is only when, in addition to the local and general reaction, we get the focal also that the test tells us where is the site of the disease.

In patients who have been formerly treated with tuberculin and who still have a high degree of immunity, local reactions may occur with only moderate doses, doses which are far too small to evoke general let alone focal reactions. In fact the occurrence in such patients of local reactions alone is a good sign.

Although doubtful reactions, as described above, sometimes occur, they are unusual, and if the precautions described be adopted there is a most striking contrast between the complete absence of any slightest sign local, general or focal of reaction in those who have no tuberculosis, and the very evident reaction in the morbid, so that interpretation, at least in patients who have not previously received injections of tuberculin, is easy. The local reaction shows that infection has occurred, the general reaction shows that it is still active, and the focal reaction shows where.

For patients who have been successfully treated with tuberculin and require to be tested six or twelve months later to exclude relapse, larger and more numerous test doses are needed, as 0·005 cubic centimetre, 0·015, 0·04, 0·07 and 0·11 cubic centimetre at intervals of two or three days.

It should be carefully noted that the degree of the reaction does not indicate the severity of the disease; rather the earlier and slighter the infection, the more sensitive is the patient likely to be.

The test doses recommended above are: 0·0001 cubic centimetre, 0·0005 and 0·001 cubic centimetre for a two years' baby; 0·0003, 0·0015 and 0·003 cubic centimetre for a child of six years; 0·0005, 0·0025 and 0·005 cubic centimetre for a child of ten years; 0·001, 0·005 and 0·01 for an adult.

Some may think them too large, but very many suspects, both (i) patients suffering from other diseases resembling tuberculosis, as hydatid or cancer of the lung, chronic nasal infection with chronic bronchitis, sarcoma of a joint or of glands, and (ii) uninfected kinsmen of consumptives have been found to tolerate these doses without any sign of reaction and without suffering any harm, early or late. Reaction, usually not severe, occurs only in the infected; even when severe, so as to be disagreeable or even distressing for a few days, it does no harm; rather it initiates healing.

Finally, test injections of tuberculin, an extract of the toxins of the bacillus and not the bacillus itself, do not confer immunity from future infection.

GUY GRIFFITHS, M.D.,
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British Medical Association News.

ANNUAL MEETING.

THE ANNUAL MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Darling Building, University of Adelaide, on June 25, 1931, DR. C. E. C. WILSON, the President, in the chair.

Annual Report of Council.

The annual report of the Council was received and adopted. The report is as follows.

Election.

At the annual meeting held last June the following were elected:

President: Dr. C. E. C. Wilson.
Vice-Presidents: Dr. A. V. Benson and Dr. R. E. Magarey.
Honorary Medical Secretary: Dr. Allan D. Lamphee.
Honorary Treasurer: Dr. W. A. Verco.
Members of Council: Dr. J. B. Birch, Dr. R. G. Burnard, Dr. E. Britten Jones, Dr. F. L. Wall. (Dr. P. S. Messent and Dr. F. St. J. Poole remained for another year.)
Federal Committee: Sir Henry Newland and Dr. Bronte Smeaton.

At the first meeting of the Council, held on 3rd July, 1930, the following subcommittees were appointed:

Scientific: The President, Sir Henry Newland, Dr. A. V. Benson, Dr. E. Britten Jones, Dr. W. A. Verco (Honorary Secretary, convener).
Contract Practice: The President, Dr. H. Gilbert, Dr. F. St. J. Poole, Dr. F. L. Wall (Lay Secretary, Convener).
Revision of Rules: The President, Sir Henry Newland, Dr. Bronte Smeaton (Lay Secretary, Convener).
Parliamentary Bills: The President, Dr. A. V. Benson, Dr. F. St. J. Poole (Lay Secretary, Convener).
Library: The President, Sir Henry Newland, Dr. P. S. Messent, Dr. R. E. Magarey (Honorary Secretary, Convener).

Meetings.

Council.—The Council met on thirteen occasions, the attendance being:

Dr. A. V. Benson ...	10	Dr. P. S. Messent ...	9
Dr. J. B. Birch	10	Sir Henry Newland .	11
Dr. R. G. Burnard ..	12	Dr. F. St. J. Poole ..	12
Dr. H. Gilbert	11	Dr. B. Smeaton	11
Dr. E. Britten Jones	12	Dr. W. A. Verco ...	10
Dr. A. D. Lamphee .	13	Dr. F. L. Wall	12
Dr. R. E. Magarey ..	11	Dr. C. E. C. Wilson ..	13

Scientific Subcommittee.—The Scientific Subcommittee met three times, the attendance being:

The President	3	Dr. Allan D. Lamphee	3
Dr. A. V. Benson ...	2	Sir Henry Newland ..	1
Dr. E. Britten Jones .	3	Dr. W. A. Verco	1

Contract Practice Subcommittee.—The Contract Practice Subcommittee met on four occasions, including two conferences with the United Friendly Societies' Council, the attendance being:

The President	4	Dr. F. St. J. Poole ...	4
Dr. H. Gilbert	3	Dr. F. L. Wall	4

Parliamentary Bills Subcommittee.—The Parliamentary Bills Subcommittee met once, the attendance being: The President, Dr. F. St. J. Poole; and at the invitation of the Council, Dr. P. T. S. Cherry, Dr. H. Gilbert, Dr. H. Halloran, Dr. Frank S. Hone, Dr. F. W. A. Ponsford.

Library Subcommittee.—The Library Subcommittee met once, the attendance being: The President, Dr. Allan D. Lamphee, Dr. R. E. Magarey, Dr. P. S. Messent, Sir Henry Newland.

Monthly General.—Nine meetings were held during the year, three of these being clinical evenings, two of which were held at the Adelaide Hospital and one at the Children's Hospital. The attendances throughout were satisfactory. During the year it was decided to hold one of the meetings in a country centre, and Clare was chosen for the October meeting.

The following programme was carried out:

1930—

July: Clinical evening at Adelaide Hospital.

August: Paper by Dr. Jean Macnamara, of Melbourne, on "The Treatment of Acute Anterior Poliomyelitis with Human Immune Serum."

September: Paper on "Tuberculosis of the Lungs and Bronchial Glands in Children," by Dr. R. Thorold Grant.

October: Papers by Dr. J. Riddell and Dr. P. T. S. Cherry on "The Treatment of Difficult Labour in General Practice" (meeting held at Clare).

November: Papers by Sir Henry Newland on "Surgical Conditions at the Base of the Skull," and Dr. Leonard Lindon on "Some Notes on Neuro-Surgery."

1931—

February: Discussion on "Unusual Bone Conditions."

March: Clinical evening at Children's Hospital.

April: Papers by Dr. H. C. Nott and Dr. H. A. McCoy on "The Value of X Rays in Diagnosis."

May: Clinical evening at Adelaide Hospital.

Federal Committee.

Sir Henry Newland and Dr. Bronte Smeaton represented the Branch on the Federal Committee, both attending the meetings held at Sydney, October 2, 1930, and Melbourne, March 27, 1931.

Representation on Boards.

Nurses' Registration Board.—Dr. F. Steele Scott having resigned as the Association Representative on the Board, owing to ill health, in August last, Dr. S. R. Burston was appointed in his place.

Membership.

The membership of the Branch is 387. The number of new members elected was three, the balance representing the difference between transfers "in and out" after deducting deaths *et cetera*. It is with deep regret that we record the deaths of Dr. A. N. Krakowsky, Dr. A. H. Bennett and Dr. W. H. Russell.

Listerian Oration.

The Council invited Sir Charles Martin to deliver the Listerian Oration for 1931 in May. Sir Charles found, however, that it had been arranged for him to visit Queensland in connexion with his work at this time, and the Council decided to alter the date of the oration to Wednesday, June 24, so that he would be able to deliver it.

New Model Lodge Agreement.

Negotiations have been carried on throughout the year, and it is pleasing to report that finality is now being reached. The difficulties in arriving at a satisfactory position in regard to lodge work have been largely increased owing to the existing financial conditions, which members will appreciate, and the new agreement has had the earnest consideration of the Council throughout the year.

Arrangements have now been completed with the United Friendly Societies' Council, representing up to the present approximately 200 lodges, and the I.O. Rechabites, Albert District, No. 83, representing 170 lodges. One agreement will be signed by these bodies on behalf of the lodges represented by them, and the Council of the Association will sign on behalf of those members who have given them authority to do so. Lodge surgeons will be fully advised of the position in due course.

Adelaide Permanent Post-Graduate Committee.

The results of the work of the Committee have been very encouraging, the attendances at the refresher course from May 26 to 31 last year being most satisfactory. This year the Committee arranged a refresher course from May 25 to 30, including two lectures by Mr. H. B. Devine, of Melbourne, and the interest in the course has been well maintained.

Library.

Members are again reminded of the facilities available to them as members of the British Medical Association. The medical library is at their disposal, and books and periodicals may be borrowed on certain conditions. It was felt by the Council that country members were not generally aware of the fact that the advantages of the library were open to them, and in February last a circular was sent out, drawing the attention of country members to the library, and that upon payment of an extra 10s. *per annum* they could enjoy the same privileges as city and suburban members.

Work of Sections.

The Eye, Ear, Nose and Throat Section.—The Eye, Ear, Nose and Throat Section has held eight meetings during the year, the attendances being satisfactory. The section consists of fifteen members.

The Section of Clinical Medicine.—Two meetings were held, which were well attended. The membership totals 52.

Section of Anæsthetics.—This subsection was formed during the year, and consists of 18 members. Four meetings have been held.

Primary Fellowship Examination, Royal College of Surgeons.

Advice was received in July last from the College of Surgeons of Australasia that arrangements had been made for the Primary Fellowship Examination of the Royal College of Surgeons to be held in Melbourne in August or September, 1931, providing that not less than twenty-five eligible candidates are desirous of being examined.

Poliomyelitis.

At the invitation of the Council, Dr. Jean Macnamara, of Melbourne, addressed members at the August monthly meeting on "The Treatment of Acute Anterior Poliomyelitis with Human Immune Serum." Following this, a Permanent Committee has been formed, consisting of representatives of the British Medical Association and the Red Cross Society, and called the "South Australian Joint Committee for Poliomyelitis." Although only recently formed, the Committee has been actively engaged, and good work has been accomplished.

Jubilee of New South Wales Branch.

The commemoration of the fiftieth anniversary of the New South Wales Branch took place on Friday, October 3 last, when the new house of the Branch was officially opened. Congratulations were sent on behalf of the South Australian Branch, and at the invitation of the Council Sir Henry Newland represented the Branch at the function.

Australasian Medical Congress, Fourth Session.

The Fourth Session of the Congress will be held at Perth in September, 1932, and suggestions from the Council for the appointment of presidents and vice-presidents for the various sections have been forwarded.

Group Representative on the Central Council.

Dr. T. P. Dunhill was nominated for a further term of three years from next July to represent the Branch as Group Representative for South Australia, Tasmania, Victoria and Western Australia.

Annual Meetings of Parent Association, 1931.

The annual meetings will be held at Eastbourne in July, and Dr. W. Anstey Giles and Dr. J. W. Yeatman have been appointed by the Council to act as delegates of the Branch.

Medical Officers to the Destitute Board.

Owing to the existing financial conditions, the work of the medical officers to the Destitute Board has greatly increased during the year, and the Council felt that the remuneration paid to the officers was quite inadequate for the work performed. A deputation was appointed to wait on the Children's Welfare and Public Relief Department, with a view to increasing the remuneration paid and endeavouring to get the Department to take over the supply of medicine. Although the efforts were not successful, as a result of the representations made it is confidently expected that when the appointments are next being considered at the end of June, better conditions will be given to the medical officers.

Federal Council.

At a special general meeting of the Branch held on March 19 last, the Articles of Association and By-Laws of the Federal Council were approved and adopted. The Federal Council, a corporate body, will replace the Federal Committee, which has been in existence for eighteen years, and will be able to more effectively carry out the objects of the British Medical Association on behalf of the various Branches.

Farmers' Relief Act.

As a result of the efforts of the Council, the Government has given medical practitioners a "second preferential claim" under the Act. This should afford a measure of relief to country members and prove more satisfactory than the provisions of the previous acts, which did not recognize any claim for medical attendance.

Building for the Branch.

No steps have been taken in regard to a building for the Branch during the year, the Council considering that the time was not opportune to proceed in this direction.

Gifts to the Branch.

During the year the following gifts were made to the Branch: Dr. T. G. Wilson, five shares in British Medical Hall Company, Limited, and one debenture in the Australasian Medical Publishing Company, Limited, and Dr. A. A. Lendon, one share in the British Medical Hall Company, Limited. The Council greatly appreciates these gifts, and thanks the donors for their generosity.

(*Signed*) C. E. C. Wilson,
President.

Financial Statements.

The financial statements, as submitted by the Honorary Treasurer, Dr. W. A. Verco, were received and adopted. The statements are published herewith.

Income and Expenditure for Year ended December 31, 1930.

	£ s. d.	£ s. d.
To British Medical Association, London	424 10 0	
„ British Medical Association, London—Unpaid Subscriptions	62 9 6	
		486 19 6
„ The Medical Journal of Australia	411 7 0	
„ The Medical Journal of Australia—Unpaid Subscriptions	61 5 0	
		472 12 0
„ Lister Medal		0 10 0
„ Telephone	25 13 4	
„ Exchange	1 4 4	
„ Rent	54 0 0	
„ Postages and Telegrams	34 4 6	
„ Stationery and Printing	51 2 6	
„ General Expenses	86 12 9	
„ Advertising	0 15 0	
		253 12 5
„ Salary		350 0 0
„ Federal Capitation Grant		39 14 0
„ Depreciation		14 16 8
„ Balance		46 2 8
		£1,664 7 3

	£ s. d.	£ s. d.
By Medical Certificates		0 4 9
„ City Subscriptions	967 7 0	
„ Country Subscriptions	472 9 6	
		1,439 16 6
„ Accrued Subscriptions		204 6 0
„ Interest		20 0 0
		£1,664 7 3

Library Fund Account, December 31, 1930.

	£ s. d.	£ s. d.
To University of Adelaide Library Grant	50 0 0	
„ Balance carried down	518 12 1	
	£568 12 1	
By Balance brought down December 31, 1929		453 5 5
„ Savings Bank Interest		5 6 8
„ Transfer from General Fund, 219 full at 10s. and 2 half subscriptions at 5s.		110 0 0
		£568 12 1

General Fund Account.

	£ s. d.		£ s. d.
To Library Fund Account	110 0 0	By Balance brought down, December 31, 1929	2,800 4 4
„ Balance	2,736 7 0	„ Amount Transferred from Income and Expenditure Account, December 31, 1930	46 2 8
	£2,846 7 0		£2,846 7 0

Balance Sheet as at December 31, 1930.

LIABILITIES.	£ s. d.	£ s. d.	ASSETS.	£ s. d.	£ s. d.
To Subscriptions paid in advance		5 4 0	By Plant and Fittings	148 6 9	
„ British Medical Association, London	511 4 0		*Less* Depreciation	14 16 8	
„ THE MEDICAL JOURNAL OF AUSTRALIA	61 5 0				133 10 1
		572 9 0	„ Investments—		
„ Library Fund		518 12 1	Commonwealth Loan (5%, 1948)	403 12 0	
„ Sundry Creditors		3 4 4	British Medical Hall Company, Limited	1,850 0 0	
„ General Fund		2,736 7 0			2,253 12 0
			„ Library Fund—		
			Delineascope	55 2 10	
			Kodascope	79 10 0	
					134 12 10
			„ Lister Medals and Dies		20 13 4
			„ Savings Bank (Library Fund)		118 14 3
			„ British Medical Hall Company, Limited—Loan		450 0 0
			„ Subscriptions owing		241 14 0
			„ Stocks—		
			Medical Certificate Books	4 11 3	
			Hospital Forms	0 19 1	
					5 10 4
			„ Sundry Debtors		8 0 0
			National Bank of Australasia, Limited	2 9 8	
			Savings Bank	450 0 0	
			In Hand	16 19 11	
					469 9 7
		£3,835 16 5			£3,835 16 5

W. A. VERCO, Honorary Treasurer.
WALTER C. DOBBIE, Lay Secretary.

Audited and found correct according to the books and vouchers produced.

MUECKE, WILTSHIRE & Co.,
Chartered Accountants (Australia),
Auditors.

Adelaide,
May 30, 1931.

Election of Office-Bearers.

Officers for the ensuing twelve months were elected as follows:

President: Dr. A. V. Benson.
Vice-President: Dr. F. St. J. Poole.
Honorary Medical Secretary: Dr. A. D. Lamphee.
Honorary Treasurer: Dr. W. A. Verco.
Members of Council: Dr. P. Gorrie, Dr. H. Powell.
Delegates to the Federal Committee: Sir Henry Newland, Dr. Bronte Smeaton.

President's Address.

DR. C. E. C. WILSON, the retiring President, delivered his President's address (see page 469).

Correspondence.

PUERPERAL SEPSIS.

SIR: In a discussion on puerperal sepsis following three papers which had been read before the Victorian Branch of the British Medical Association, a list of various treatments which one speaker had used and found wanting included antidiphtheritic serum. He did not say whether he had administered it orally or hypodermically, and in the treatment of *Streptococcus hæmolyticus* it all depends on the method of administration. Orally used, the serum definitely fails to treat *Streptococcus hæmolyticus* satisfactorily. Hypodermically it has not failed to rapidly clear up the cases in which that organism is found.

Orally the specific antitoxin is not absorbed and therefore can be of no value. Orally the endocrine antitrypsin serum content is absorbed and produces an action determined by the hormone development in the animal necessary to protect its tissues from the toxin administered to produce immunity. This is exactly similar to the absorption of preparations of dead glands, except that the hormone balance is produced by the living glands and is not the crude product of a laboratory. Thus the result is to stimulate the tissues to resist the toxin and to render them refractory to the infection. This action is capable of efficiently replacing all tissue stimuli which may be indicated in the case and is available for tissue stimulation in the presence or absence of any infection or locally for the antiseptic healing of wounds or burns.

Hypodermically the hormone content is not available for tissue stimulation, but the specific antitoxin provides a specific action on toxin and organism. I am a firm believer in the absolutely specific character of diphtheria antitoxin. The toxin contains two distinct and separate entities—a paralysing and a proteolysing potency. The antitoxin, being specific, must necessarily provide an antitoxin to each of these elements. No specific single biochemical reaction could cover two such differentiating entities. So that diphtheria antitoxin must contain at least these two separate products of the reaction to toxin. The paralysing element represents the streptococcus, another proteolysing the staphylococcus. And the thousands of case successfully treated confirm this conclusion.

Hence orally we obtain the endocrine antitrypsin action and hypodermically the specific antibodies to the two organisms mentioned.

Those of us long enough in practice remember the terrible days we had with diphtheria before antitoxin arrived. Sepsis was a constant complication, as it still is in so many diseases, but where today are the septic complications in diphtheria which has been efficiently treated by the hypodermic injection of the antitoxin? They do not occur. Without realizing it, we have had forty years of definite proof of the specific character of the action of diphtheria antitoxin on the septic organisms. That action alone accounts for a considerable percentage of the reduced mortality from the disease.

For its action by hypodermic injection in the treatment of *Streptococcus hæmolyticus*, I may mention that a friend of mine treated forty cases of scarlet fever with one injection of the antitoxin—dose 4,000 units. Results were throats cleared up in two days, temperature fell to normal in three days, no septic complications and no deaths. The rash was the only thing uninfluenced by the treatment. A recent case was that of a varicose ulcer in the ankle which had become infected (streptococcus) and which had involved the greater part of the tarsus. The patient had been vigorously treated orally with diphtheria antiserum which had benefited it materially, but had failed to prevent its advance. One injection of 4,000 units brought the whole process to an end in forty-eight hours and recovery was uneventful.

It has invariably been my practice, when the oral use of antidiphtheritic serum in septic cases is not giving the results expected (and I do not remember this to have happened in the presence of the staphylococcus), to add a hypodermic injection and always with the happiest result.

I have only known of one case of staphylococcus infection, a long-standing furunculosis, which required a second injection, but the second was successful (used in a test with no previous oral use of serum).

One case of scarlet fever dying, even when antidiphtheritic serum had been efficiently inhibited, was reported to me by a friend. I shall be interested to hear how the antiserum was administered and failed to act.

Yours, etc.,

D. Montgomerie Paton.

Kilmore,
Victoria,
September 21, 1931.

THE MIND.

Sir: The mind's prelude to adventure into the unknown is necessarily obscure and incomplete, and what I now write is but prelude. As I am in my seventy-fourth year I leave the adventure to those who follow.

White light defines an object with equal power, whether the seeing eye is normal or blind to red and green. Blue blindness is very rare, so rare that in many thousands of examinations I detected only one case. The effect of blindness to red and green has an effect in many cases quite obvious in certain emotional appeals. It finds expression in the face and in the voice. There is a marked limitation, both in the tone of a voice and the expression of a face, as compared with that of a unit of full tone and colour. Full tone and colour units are, however, comparatively rare. In a proportion of normal units there may be little beyond ability to recognize and name colours correctly, whereas others are gifted with a keen appreciation of colour. There may, however, in both the colour-blind and the colour-normal, be an equal appreciation of shade. In my experience that condition is at least as keen in the colour-blind as in the colour-normal.

When a writer has expressed his idea, either in words or musical form, his thought is dormant, the energy—the appeal—concealed in his work is latent until thought is stirred through his script in the reader or listener. The groups of nerve cells, involved in the writing of this script will be more or less sympathetically incited into action in the reader or listener. The inherited conditions of receptivity in the various groups of cells will, however, vary. The effect on the reader or listener may therefore be indifference, sentiment of pleasure in varying degrees. In two highly trained and, from an examination standpoint, alert and normal minds of apparently equal value there may be a complete difference of opinion as to the script's appeal. Why?

All the literature of life, whether the words of a writer or the notes of a musician, is created through the action of nerve cells, expressing themselves either in written words or in sound, and whether the appeal be through sight or hearing, a marked difference in its valuation by individual units arises, due to an emotional element—excluding scientific precision—colouring the appeal. May not, it is suggested, a thought ray contain many wave lengths, one of which is an inherited dominating factor—in units capable of appraising the appeal—though not necessarily a dominating factor, in the unit producing it, and may not such a ray suffer refraction when entering the brain, each wave length being deflected to a specialized group of cells? If the reader will admit that a normal perception of red and green has a real influence upon the range of expression in the face and voice, would not a composite appeal which involved, although concealed, a sensation of colour, furnish a reasonable explanation. Every human unit, experts state, can be identified from every other by the imprint of the finger tips; is it not reasonable to surmise that a mixed appeal involving an emotional element would, if as definite a method of appraisement as in finger tip identification could be formed, show at least as marked a distinction? At least it raises the interesting speculation that all sight and all sound registers itself in the mind, not with unvarying precision, but with infinite variety. What is exquisite to one is nought to another, all human concepts being strained through the sieve of individuality.

Yours, etc.,

George Henry Taylor.

Mosman, New South Wales,
September 22, 1931.

THE TREATMENT OF WARTS.

Sir: I have noticed the treatment of warts in your journal of September 26, and although I have no doubt they can be cured by the X rays in this manner, yet I would like to bring under notice a less elaborate and much easier method that I discovered, which is very effective to any one possessing a high frequency or diathermy apparatus. It is done with the high frequency spark, and quite painlessly, requiring no anæsthetic. The electrode is a bundle of about twenty fine iron wires soldered at one end and fastened to a suitable holder. One must avoid actual sparking or the patient will not stand it, in the following way. Lay the wires along the surface, pressing firmly to make a good electrical contact, the end of the wires on the centre of the wart, then gently raise it. There will be no sensation until it is upright; a slight warming then commences, and if the pressure is slowly relaxed a poor contact is made, and in half a minute the wart is devitalized. Then shut off the current. The electrode must not be taken away at any time when the

current is on, or a spark crosses, which makes the patient nervous. I have often used it on nervous women without any difficulty. A small current only is required. It is specially useful in small multiple warts. A single large wart is just as easy, but requires longer contact, one or even two minutes after previously moistening with 5% carbolic lotion is then quite painless. I have never known it to fail.

Yours, etc.,

T. G. BECKETT.

Melbourne,
September 27, 1931.

Books Received.

THE STARS IN THEIR COURSES, by Sir James Jeans, M.A., D.Sc., Sc.D., LL.D., F.R.S.; 1931. Cambridge: The University Press; Sydney: Moore's Book Shop. Post 8vo., pp. 198, with illustrations. Price: 8s. 6d. net.

ENGLAND'S CRISIS, by A. Siegfried, translated by H. H. and D. Hemming; 1931. London: Jonathan Cape; Sydney: Moore's Book Shop. Demy 8vo., pp. 256. Price: 17s. 6d. net.

Diary for the Month.

OCT. 20.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
OCT. 23.—Queensland Branch, B.M.A.: Council.
OCT. 27.—New South Wales Branch, B.M.A.: Medical Politics Committee.
OCT. 28.—Victorian Branch, B.M.A.: Council.
OCT. 29.—South Australian Branch, B.M.A.: Branch.
OCT. 29.—New South Wales Branch, B.M.A.: Branch.
NOV. 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.
NOV. 5.—South Australian Branch, B.M.A.: Council.
NOV. 6.—Queensland Branch, B.M.A.: Branch.
NOV. 10.—New South Wales Branch, B.M.A.: Ethics Committee.
NOV. 11.—Victorian Branch, B.M.A.: Branch.

Medical Appointments.

Dr. W. I. Clark has been appointed to the Mental Deficiency Board as a Medical Officer of the Department of Public Health, Tasmania, pursuant to the provisions of Section 39 (2), 1, of the *Mental Deficiency Act*, 1920.

* * *

Dr. A. N. Magnus (B.M.A.) has been appointed Medical Officer of Health by the Perth Road Board, Western Australia.

* * *

Dr. D. H. E. Lines (B.M.A.) has been appointed President of the Medical Council of Tasmania.

* * *

Dr. W. W. Giblin (B.M.A.) has been appointed a Member of the Medical Council of Tasmania.

* * *

Dr. J. E. F. Stewart (B.M.A.) has been appointed to act as Medical Inspector of Seamen at Carnarvon, Western Australia, pursuant to the provisions of Section 123 of the *Navigation Act*, 1912-1926.

* * *

Dr. C. Joyce (B.M.A.) has been appointed to act as Medical Inspector of Seamen at Onslow, Western Australia, pursuant to the provisions of Section 123 of the *Navigation Act*, 1912-1926.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes*, sought, etc., see "Advertiser," page xiv.

LAUNCESTON PUBLIC HOSPITAL, TASMANIA: Medical Superintendent.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members desiring to accept appointment in ANY COUNTRY HOSPITAL, are advised to submit a copy of their agreement to the Council before signing, in their own interests. Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.